

# THE MEDICAL CLINICS OF NORTH AMERICA

---

VOLUME 3

NUMBER 5

---

CONTRIBUTION BY DR. JOHN B. DEAVER

LANKENAU HOSPITAL

---

## CHRONIC APPENDICITIS

THERE is a general impression that if there is one subject on which there is little that is new to be said it is appendicitis. Indeed, the impression is a perfectly correct one, but the circumstance remains that among so many of the profession at large the facts concerning appendicitis are either not understood or ignored that they must be repeated again and again in order that their importance may be more universally recognized. It is for this reason that I have yielded to the flattering persuasion of the editor to appear in the Medical Clinics of North America.

When the character of appendicitis in its acute form is better understood it is probable that the chronic type will be less frequently seen than it is at the present. For it is known that many cases of chronic appendicitis are based on previous acute attacks in which spontaneous improvement has taken place or which have yielded to rational treatment, consisting of complete physiologic and anatomic rest, ice-bags, etc. This candid admission that acute appendicitis may yield to treatment or may subside by absorption, however, presents only one aspect of the subject, for it is a well-known fact that a very large percentage of cases of acute appendicitis go on to peritonitis and abscess formation, and no one can foretell the outcome of these cases except the surgeon at the operating table. Whether the verdict is to be a favorable or unfavorable one depends not so much on the skill of the surgeon as on the intuition or judgment of the

physician in attendance in seeking surgical advice at the most favorable stage of the disease. In other words, whether the case is seen early, before peritonitis has had a chance to develop, *i. e.*, within twenty-four to forty-eight hours, or whether in the early or late stage of peritonitis. A word of warning may not be out of place right here with regard to the cases seen on the second or third day or later, and which seem to be subsiding. It must be remembered that gangrene has an insidious way of developing under the appearance of a temporary subsiding of symptoms, and also that perforation of the appendix takes place without any premonitory symptoms. These items, together with the fact that by the end of the second day a diffuse peritonitis may possibly have developed, present the strongest arguments in favor of seeking surgical advice within the first twenty-four to thirty-six hours of a case with acute abdominal symptoms. I say "acute abdominal symptoms," for the differentiation of appendical and other acute abdominal diseases is not always easy or simple even to the experienced practitioner, be he surgeon or internist. But I have no hesitancy in saying that, on the whole, the surgeon is in a better position to make the diagnosis for the simple reason that he sees more pathology than does the internist, and thus is better able to correlate the revelations of the operating table with the picture at the bedside.

In a case of death following acute appendicitis how often does the death notice read, "the result of operation for appendicitis"; how much more truthful if it read, "death from a belated operation." Unhappily the three words—appendicitis, operation, and death—are thus connected in the public mind. If the surgical treatment could always be quick there would be little chance for death. If the physician would say, This is appendicitis and not a case for me, but for the surgeon, there would be much less loss of precious time and much less loss of life. This applies to chronic appendicitis as well. If the surgical treatment could always be quick there would be little chance of death.

An acute abdominal affection ought not to be considered as a medical case. Chronic abdominal affections, not primarily medical, are the property of the surgeon. Meet the enemy at the



gate and exterminate him before being given the opportunity to distribute vicious propaganda (micro-organisms) and form dangerous and entangling alliances with the viscera in the immediate as well as those distant to the region primarily attacked.

The puzzling thing about appendicitis is its protean character. This is especially true of the chronic disease. With the more general recognition that chronic appendicitis may simulate any one of the diseases of the abdomen, not excluding genito-urinary and pelvic disorders, there is no doubt that much less unnecessary surgery will be done. Most commonly the disguise is that of some disease of the upper abdomen, particularly cholecystitis and duodenal or gastric ulcer. Various concise terms, such as appendicular gastralgia or appendical dyspepsia, have been suggested to designate this deceptive type of chronic appendicitis, but I prefer to call it "appendicitis with referred symptoms," which, while less concise a term, is at once descriptive and comprehensive.

Furthermore, there is the fact of the frequent association of chronic appendicitis with one or another disease of the abdomen, which leads to the still moot question as to cause and effect. Not a few authorities are inclined to believe that cholecystitis, for example, is the cause of appendicitis (Dieulafoy), while others attribute the two to a common cause (Lane). Personally I have no reason to change the opinion so frequently expressed that the basic factor is infection residing in the appendix and from there extending to the other abdominal viscera. This conception tallies entirely with modern theories of focal infection. And the impression is furthermore strengthened by the fact that in so many cases of cholecystitis and of peptic ulcer that come to operation the appendix is present and nearly always diseased, if not in its gross appearance, at all events, microscopically. I feel reasonably confident also that in peptic ulcer, where the appendix has been previously removed, inspection at the first operation would have then likely, at that time, have demonstrated the ulcer.

The **diagnosis** of chronic appendicitis is, therefore, not one that can be hastily made as far as surgical interference is con-

cerned. The importance of the first-hand information to be gained from a carefully taken history and a careful physical examination has in no way been superseded by modern aids to diagnosis, useful and valuable as these are. Practically every case of chronic appendicitis gives a history of pain and some sort of digestive trouble. There is also some tenderness and rigidity in the right iliac fossa and a feeling of soreness, especially after taking exercise. In addition to these, the more prominent local symptom, constipation, often forms part of the history; it gives rise to general symptoms of toxemia, affecting the nervous circulatory and muscular systems, and presenting the picture to which we apply the convenient term "neurasthenia." Morris has aptly traced the effects of the constant irritation of the constipation on certain sympathetic ganglia, producing a sort of languor in the muscular coat of the bowel; the resulting auto-intoxication leads to the pathologic changes in the brain, the suprarenals, and the liver, which account for the neurasthenic symptoms which form part of the history of so many cases of chronic appendicitis.

In some instances the condition passes on to a true bacterial infection, such as rheumatoid arthritis. This fact was forcibly impressed upon me a few years ago when I was called to see a patient, a woman of middle age, who was already in a moribund state from peritonitis due to a ruptured appendix. The history of the case showed that the patient had suffered for several years from rheumatism of the joints, especially at the knees, and that previous to this she had had occasional attacks of pain in the abdomen referred to the epigastrium and the lower right quadrant, with occasional soreness in the latter region. Constipation was also part of the history. She consulted a renowned physician in a metropolis in the middle west, a man greatly interested in the subject of focal infection and the administration of autogenous vaccines. She remained under his care for some time, and was finally dismissed as practically well, and advised to go home. When she returned to her home in Philadelphia she was seized with the above attack which caused her death. This woman in all likelihood had a chronic appendicitis, and if at the

time of consulting the aforementioned physician (or before that) she had had her appendix removed, she would probably have been relieved of her rheumatoid affection, and might be living today.

Turning to some of the visceral diseases with which appendicitis is commonly confused and oftentimes associated, they are duodenal ulcer, gastric ulcer, and cholecystitis, with or without calculus, and renal as well as pelvic disorders.

The typical duodenal ulcer, as is well known, gives a history of periodicity of attacks of pain and indigestion and a definite time-relation to food-intake. But often such a history is wanting and no decision can be made without resort to x-ray, gastric analysis, etc. The x-ray sometimes does and sometimes does not indicate ulcer, even when the latter is present, but it is useful in demonstrating the presence of stone or other foreign body in the appendix.

Although gastric analysis is not always reliable, the stomach contents should be examined for the completeness of data, and for the information that may possibly be derived therefrom. In doubtful cases we turn to every diagnostic aid at our disposal. And even after these have been exhausted, we fail frequently enough in our conclusions. In fact, with the abdomen open the surgeon is not always sure, and how often, especially in gastric cases, is he obliged to open the stomach before he feels able to make a diagnosis. Pylorospasm is often present in peptic ulcer and sometimes in appendicitis. When present there is usually a delay in the passage of food through the stomach and hyperacidity, but often the reverse is true—hypermotility and hypo-acidity—variations that also occur in ulcer cases. Moreover, abnormal gastric findings may not necessarily be due to gastric disease or impairment of gastric function, but may be the effect of disease elsewhere, notably the lower intestinal tract. As a general rule it may be said that the more difficult it is to determine the cause of chronic indigestion, the greater the likelihood that it resides in the appendix. It is my experience also that the appendix that contains stone or stones is the one that is more apt to give rise to symptoms referred to the epigastrium;

the symptoms in this type of chronic appendicitis and in the fibrous and obliterated, etc., being due to reflex action.

In gall-bladder disease the differentiation is oftenest made on the character of the pain, which in calculous cholecystitis is colicky and in the non-calculous disease is more apt to be referred to the shoulder and back. But the very frequent association of the two diseases, undoubtedly the result of ascending infection, makes differential diagnosis all the more difficult.

The differentiation between chronic appendicitis and renal conditions, especially stone in the ureter, is generally not possible except with the aid of the *x*-ray, the cystoscope, or ureteral catheterization. The confusion of symptoms in such instances is generally traceable to variations in the position of the appendix—a retrocecal appendix. But when there is no such close proximity of the renal organs and the appendix, the symptoms may be due to retroperitoneal inflammatory processes or secondary changes from lymphatic involvement from a diseased (infected) appendix. Abnormal position of the appendix also accounts for pelvic symptoms and pelvic disease which so often mimic and accompany appendicitis.

The high position in which the appendix is so frequently placed is, without doubt, responsible for many errors in differentiation of upper abdominal conditions. Another fruitful source of error is the presence of adhesions. It is a well-known clinical fact that adhesions may simulate appendicitis and vice versa, but it is my experience that adhesions in the upper right abdominal quadrant are more frequently caused by chronic appendicitis than otherwise.

Confusion in diagnosis is not infrequently caused by the presence of cecum mobile, tubercular adenitis of the terminal mesenteric glands, and enteritis, in which removal of the appendix is not indicated unless it is diseased. Some cases of gastro-enteroptosis with tenderness in the cecal region also often suggest a chronic appendicitis. This diagnosis is generally possible with the *x*-ray, but I have no hesitancy in saying that removal of a diseased appendix in these cases is of undoubted benefit.

Another condition in which I believe removal of the appendix

to be indicated is in certain instances of colitis, in which the appendix plays a rôle without a doubt. But in order to be of benefit the appendectomy should be done before extensive infiltration of the walls of the colon has taken place. Once this has occurred not much good can be expected from the operation.

Early carcinoma of the cecum also frequently gives rise to symptoms of appendicitis. Prompt diagnosis is important because prompt operation is so essential. In this connection it may not be amiss to say a few words on the subject of primary carcinoma of the appendix, which, while not common, is by no means rare.

In a study of 13,151 cases, taken from my material at the Lankenau Hospital of Philadelphia, Reimann,<sup>1</sup> the pathologist of the hospital, found carcinoma in from 0.3 to 1 per cent. of appendices, subjected to a gross and microscopic examination. The outstanding facts of the study are: (1) The carcinomatous appendix *per se* does not present any symptoms other than those of an ordinary acute or chronic appendicitis; in fact, in some instances carcinoma was found in the appendix removed as a routine measure, without having presented any signs of appendicitis. (2) Carcinoma is not always evident on gross inspection, either *in situ* or in the pathologic laboratory, but is demonstrable on microscopic examination. (3) Although usually benign, extension and metastasis from primary carcinoma of the appendix have been reported, so that malignancy has to be reckoned with.

Carcinoma or sarcoma of the terminal ileum also, when not far enough advanced to cause symptoms of itself, can rarely be differentiated from chronic appendicitis except by incision and inspection. I have had two such instances within the past fortnight.

Appendectomy as a routine measure when operating for intra-abdominal disease is undoubtedly a justifiable and warranted procedure, and I am glad to say one which is steadily gaining in favor among surgeons. The arguments advanced against the additional operation are that it may be depriving the patient

<sup>1</sup> Amer. Jour. Med. Sci., 1918, clxvi, 190.

of an organ of whose function we are still ignorant, and that it adds to the length of the operation and increases the danger of shock. Both arguments are easily controverted. Whatever the unknown physiologic function of the appendix may be, it is one which the individual is apparently well able to dispense with, if he is not actually better off without it; and, second, shock is practically a negligible quantity in the hands of the experienced operator, since the appendix nearly always can be delivered and removed in a very few minutes.

Appendectomy is probably the safest operation in the surgeon's repertory. The mortality in uncomplicated cases of appendicitis is about  $\frac{1}{2}$  per cent. or less, the liability is minimal, and the results, beyond question, beneficial. In fact, it looms large as an important contributing factor in preventive medicine, the watchword of the profession today.

There are a number of more or less unusual conditions which we are occasionally called upon to differentiate from appendicitis, such as epigastric hernia, incipient inguinal or femoral hernia, early psoas abscess, early Pott's disease, chronic diverticulitis, aneurysm of the abdominal aorta, gastric crises of tabes, etc.

The pathologic condition of the appendix bears no constant relationship to the length of time that the symptoms of the disease have existed; therefore the medical man should never entertain the thought that there is no immediate hurry in the given case.

FROM THE MEDICAL CLINIC OF THE JEFFERSON  
MEDICAL COLLEGE AND HOSPITAL

**Low Blood-pressure**

By THOMAS McCRAE, M. D.

**Malignant Disease of the Lung**

By ELMER H. FUNK, M. D.

**Analysis of Diseases of the Gall-bladder and Ducts**

By MARTIN E. REHFUSS, M. D.

**Some Aspects of the Diagnosis and Treatment of Cholecystitis  
and Cholelithiasis**

By B. B. VINCENT LYON, M. D.

**Chronic Valvular Heart Disease**

By E. J. G. BEARDSLEY, M. D.

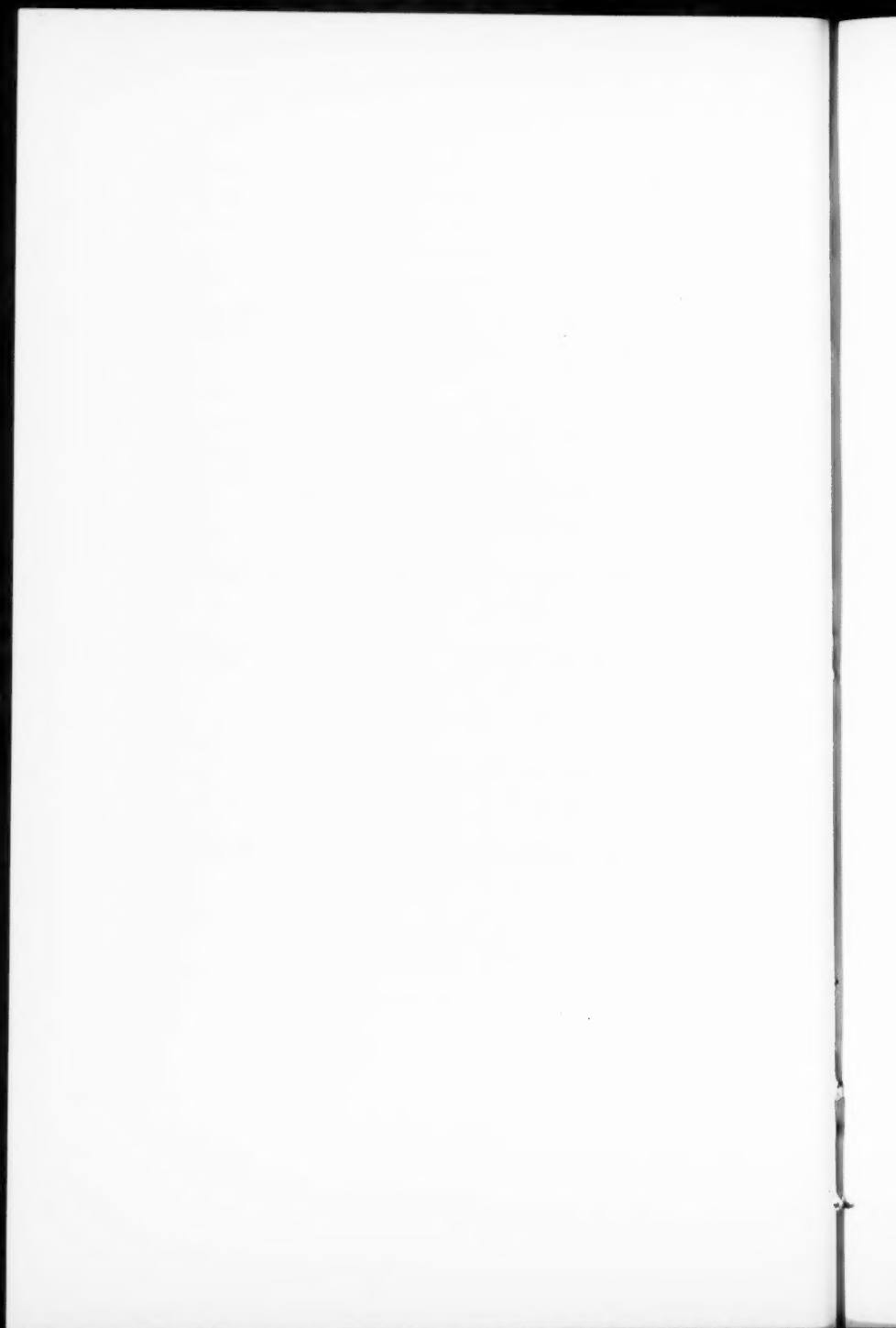
**Ethics, Ideals, and Efficiency in the Practice of Medicine**

By E. J. G. BEARDSLEY, M. D.

**Discussion of Diabetes Mellitus in Children**

By H. K. MOHLER, M. D.





## CLINIC OF DR. THOMAS McCRAE

JEFFERSON HOSPITAL

### LOW BLOOD-PRESSURE<sup>1</sup>

**Discussion of the Parts of the Circulation which May Be Responsible for Low Blood-pressure; Discussion Clinically of the Various Groups, with a Number of Illustrative Cases; Importance of Recognizing the Fact that Low Blood-pressure is a Symptom and Not a Disease; Need for Careful Study to Determine the Underlying Causes.**

As you go on in medicine you will be impressed by the fact that improvement in our means of examination, while it adds to our knowledge, adds also to the complexity of the problems which we have to solve. The improvement in the means by which we estimate blood-pressure is no exception to this. We did not have so many things to consider in the days before we had exact blood-pressure estimations. In general, we may say that high blood-pressure has received more attention and been studied much more thoroughly than low pressure. In this and some succeeding clinics I hope to bring before you examples of some of the various problems presented by hypotension.

If you come to the study of blood-pressure readings with any notion that it is a simple problem which involves little more than the statement of certain figures, please get this idea out of your minds. To state that a patient has a systolic pressure of 220 or 90 mm. Hg. gives us a fact, but a sterile fact, if we carry our investigations no further. There are some men who consider that a diagnosis is made if a patient is found to have hypertension.

<sup>1</sup> This represents a combination of clinics given at the Jefferson and Pennsylvania Hospitals.

We must remember that high or low blood-pressure is not a disease in itself, but only a symptom due to disease somewhere. It may be compared to fever for which we have always to find a cause. In some cases we may have a patient showing fever and little else, so that we are at a loss to make a diagnosis. The same happens occasionally with variations in pressure, although here I should say that the difficulty arises more often with high than with low blood-pressure. In both the explanation often lies with more than one causal factor, as we shall find later. However, we should determine first what we are going to regard as low pressure. Mr. X, what figure would you suggest at which we may say low pressure exists?

STUDENT: At 100 mm. Hg. for the systolic pressure.

DR. MCCRAE: You are certainly safe in giving that figure. Any adult with a pressure of 100 mm. comes in the low-pressure class. My suggestion is to make the figure a little higher, namely, 110 mm. Hg. for an adult. You will generally find that at or below this figure we may have symptoms of low pressure. Where would you put the figure for the diastolic pressure?

STUDENT: I do not know.

DR. MCCRAE: This is a wise answer and most men would agree with you. As you study cases of low systolic pressure due to various causes you will see that there is greater variation in the diastolic than in the systolic pressure. I find difficulty in giving any set figure. One patient shows a systolic pressure of 100 and a diastolic of 90, and another has 100 systolic and 55 diastolic. In this connection we are discussing cases with a low systolic pressure; the instances of low diastolic pressure with normal or increased systolic pressure in aortic insufficiency are not included. Before going on to the study of abnormal conditions let us first go over the factors which keep the blood-pressure at the usual figures. What are these?

STUDENT: The force of the heart itself and the condition of the arteries.

DR. MCCRAE: What factors are concerned in the latter?

STUDENT: The vasomotor system.

DR. McCRAE: What example can you give of a sudden failure of this?

STUDENT: An ordinary fainting attack.

DR. McCRAE: Correct. Some emotional disturbance suddenly upsets the control, with a resulting syncope. What factors are concerned in the proper vasomotor control?

STUDENT: The vasomotor center.

DR. McCRAE: Is that all?

STUDENT: The influence of the secretion of the adrenals.

DR. McCRAE: Up to this point the subject of the control of the vasomotor system seems comparatively easy, but now we are in deep waters at once. Any question pertaining to the disturbance of the internal secretions is a complex one. It runs through the explanation suggested in several of the forms of low pressure.

Let us discuss first the parts of the circulation which may be responsible for low blood-pressure and later take up some of the clinical examples of the various forms.

1. **Myocardium.**—Evidently weakness of this may be the cause of low pressure, but the problem is not as simple as this statement might suggest. For example, a dilated heart, which presumably has much diminished driving power, may be associated with very high or with low pressure. The former we have difficulty in explaining. Again, what we are inclined to explain as due to weak myocardial action may really be caused by vasomotor disturbance, as prolonged low arterial pressure may result in weak contractions. Certain cases of this kind stand out particularly: (a) The acute infections. Particularly in typhoid fever and pneumonia the hypotension may be marked. In typhoid fever it rarely causes any concern and sometimes is even helpful, as in hemorrhage, while in pneumonia it is generally regarded with anxiety. Some of you have seen a patient in the ward with pneumonia who on admission had a systolic pressure of 55. It may be worth noting that the rule which attaches great importance in pneumonia to the relation between the pulse-rate per minute and the systolic pressure in mm. Hg. appears to some of us to have had undue importance given to it. It is a

very uncertain guide in prognosis. I do not mean to suggest that the state of the pressure is of no value in pneumonia, but many other things have to be considered. (b) Myocarditis. This may, but does not always, give low pressure.

2. **Arteries.**—It is evident that the elasticity of the arteries has an important bearing on the maintenance of diastolic pressure particularly. With absolutely rigid arteries the pressure would fall to zero between the heart-beats. There is a very common idea that arteriosclerosis usually results in high pressure, but such is not the case; in fact, the contrary is the more common, but how much is due to the arterial changes and how much to myocardial disease may be difficult to estimate. One form of arterial disease is worthy of notice, namely, chronic aortitis. In this the pressure is low as a rule, but one would expect this to be more in the diastolic than the systolic reading. Probably changes in the small arteries are rarely responsible for low pressure. In "wound shock," which often depends on an insufficient amount of blood, it is evident that the arteries are not responsible.

3. **Capillaries.**—Here perhaps we have the seat of disturbance for many cases of both hyper- and hypotension. The chief fall in pressure as we go from the heart to the peripheral circulation occurs between the smaller arteries and the capillaries. Of many of the influencing factors we have little idea.

4. **Veins.**—These may play a part by holding large amounts of blood and so decreasing the amount of material with which the heart and arteries can work. The venous pressure influences the filling of the heart. As Cannon<sup>1</sup> has pointed out, the view that the individual often "bled into his own vessels" has been accepted on insufficient evidence, and is probably not as important as we had thought.

Back of the problem as to the situation of changes in the vascular system comes the question of explanation. For the myocardium we have some explanation in definite anatomic changes, but for the peripheral circulation we must look else-

<sup>1</sup> Jour. Amer. Med. Assoc., 1918, 70, 611.

where. Two causes probably stand out: (a) disturbed nervous control and (b) the effect of internal secretions.

5. **Blood.**—Alterations in this may be responsible for a lowered blood-pressure which may result in various ways.

(a) *Amount.*—The influence of this is seen in cases of sudden severe hemorrhage and is influenced greatly by the rate of bleeding. The removal of 5 c.c. of blood per kilogram of body weight in an animal does not influence the blood-pressure, but each subsequent similar amount causes a drop which increases in extent. The absence of effect of the removal of blood at first is probably due to vasoconstriction. In the problem of handling patients who have lost large amounts of blood the blood-pressure is an important indication. But do not imagine that the drop in blood-pressure following a hemorrhage is necessarily always serious or requires treatment. Take the case of hemorrhage in typhoid fever for example. Here a lowered blood-pressure may be distinctly advantageous, if not too great, as probably favoring coagulation. I always remember a house officer who was found pumping every drug which he thought might have any influence in raising blood-pressure into a typhoid fever patient whose pressure had fallen after an intestinal hemorrhage. The patient was in perfectly good condition and was in much greater danger from the drugs he was receiving than from the hemorrhage. Luckily, none of the drugs had the slightest effect on his blood-pressure. A good many years ago it was the custom of many to give a saline infusion after hemorrhage in typhoid fever. This was a bad practice and given up because it seemed to determine further hemorrhage in some patients.

(b) *Anemia.*—In the absence of factors which act to the contrary, this usually results in a lower pressure. Perhaps this action is due largely to the resulting lack of efficiency in the heart muscle and arteries. It may be that the adrenal glands suffer also. Anemia is often a contributing factor when other factors are primary.

(c) *Viscosity of the Blood.*—If this is decreased, as by a saline injection, the ease with which the blood passes through the vessels will be increased and the pressure lowered. This

suggests that in cases of shock, such as "wound shock," the injection of saline solution may not result in much increase in blood-pressure. This can be overcome by using solutions of various substances, such as gum acacia or gelatin. If to the saline solution enough gum acacia is added to make the viscosity of the solution about equal to that of the blood the influence of blood-pressure is about equal to that produced by the transfusion of blood. At 7 per cent. solution of acacia is a satisfactory amount for this purpose, but this contains an excess of calcium which should be removed.

(d) *Acidosis*.—That this may play a part in causing low blood-pressure seems well established, especially by the very careful study which has been made of the cases of "wound shock" in the recent war. The mechanism is probably a lowering of blood-pressure by relaxation of the cardiac and arterial musculature, with a resulting slowing of the circulation in the capillaries. As a result of this there is decreased oxidation, and so a vicious circle is set up. In "wound shock" previous starvation, marked fatigue, and a decreased amount of urinary secretion are additional factors.

It is evident that the problem may be a complex one into which many elements enter. In some cases the cause is in the circulatory apparatus itself, but in many others we have to look for something behind this. This will come out in the discussion of some of the cases.

Clinically we have various groups, some of which may be discussed in detail.

1. **Acute Infections.**—You have seen patients with typhoid fever and pneumonia who had a low pressure, and while the low pressure in this group is generally regarded as due to vasomotor paresis, some of the experimental work speaks against this, in pneumonia at any rate. But the evidence does not seem conclusive. How much part a weakened myocardium plays in comparison with failure of the peripheral circulation is usually impossible to decide. Much importance has been attached by some workers to disturbances of the adrenal glands. If an acute infection, such as typhoid fever or diphtheria, affects these



glands so that their secretion is greatly diminished, it is easy to understand a resulting lowering of pressure. It is evident that if we are to decide properly as to treatment we must have some etiology of the low pressure. If this be largely a question of toxins acting through the nervous control, evidently general measures, especially of elimination, are important. If the heart is principally concerned, then measures directed to it are essential, and in this connection the use of digitalis demands notice. We have usually considered that digitalis was not of value in such conditions in acute infections, but recent work takes an opposite view, and the use of digitalis in pneumonia has greatly increased.

What clinical evidence have we as regards therapy on this point? There is no better example than in typhoid fever. You have seen a very toxic patient with typhoid fever who on admission had a low blood-pressure—about 85 to 90 systolic and 55 to 60 diastolic. The measure which seemed to have the most effect on this was the steady employment of tub-baths. Mr. X, how do you regard this as being produced?

STUDENT: By the stimulating effect on the vasomotor system.

DR. McCRAE: There is no doubt of this so far as the evidence goes. The radial pulse after the bath is smaller and harder as compared with the soft, flabby pulse before. The blood-pressure rises on an average of 10 points, and, what is especially significant, these results are transitory and last usually for two or three hours only. Cold given in proper dosage is the best tonic we have for the peripheral circulation. You will note that I said "in proper dosage," for the use of cold must be studied as carefully as the use of digitalis.

With reference to the parts of the vasomotor system concerned it is evident that depression of the vasoconstrictor fibers may lead to low pressure. The mechanism of the vasodilator system is more obscure and there seems to be no tonic activity of the vasodilators. In general, the handling of the low pressure in acute infectious diseases is largely a matter of prevention, for when once established the usual measures are often unavailing.

These preventive measures have to be directed principally toward the elimination of toxins and the keeping up of the vasomotor tone. In this latter the use of cold is our most reliable measure. But cold has to be used as any other therapeutic measure—with judgment.

2. **Myocardium.**—This requires short mention, as you have seen a number of cases with myocarditis in which the pressure was low. But, as was mentioned before, this is not invariable, and what is apparently a weak dilated heart may be keeping up a high blood-pressure. It is in this group particularly that we find a low systolic with a relatively high diastolic pressure. Such a condition suggests a weak heart muscle. It is worthy of note that in this condition the giving of vasodilators may be useful, so that you see that a high systolic pressure is not the only indication for the use of this class of drugs. Evidently the results should be carefully observed and studied.

3. **Internal Secretion Disturbances.**—Difficult of proof as these often are, yet we must recognize that they play a part and often a large part. The problem is so closely associated with functional disturbance in the nervous system that it may be difficult to decide which is cause and which effect. In this connection the question of adrenal insufficiency is particularly important. How definite a condition is this? We know how important it seems to many of the French writers, who lay special emphasis on the "white line" as a sign of its presence. In testing for the presence of the white line it is important not to irritate the skin. It is best to draw the flat surface of the nail very gently across the skin. The upper abdomen or the lower thorax is a satisfactory area. Within a few seconds vasoconstriction results and a white line appears. This persists for a varying time. I should be glad to feel able to accept the opinions of the members of the French school who attach great importance to this sign, but the evidence is not convincing. Yet there are some cases which strongly support the view. This case, which some of you saw over a year ago, is an example of a most satisfactory result:

The patient is a single woman, aged thirty, who complained

particularly of nervous symptoms and weakness. Her past history is unimportant. For a year before her first visit she had much nervous strain with a heavy load of responsibility and anxiety. She had complained for about eight months of many nervous symptoms and very striking weakness. She has been apprehensive, feels that she has lost her usual efficiency, and has periods of depression. On getting up in the morning she usually feels dizzy for a few minutes, and bending over causes marked discomfort in the head. She states that if she lies down in the day and gets up she has the same dizziness as on getting up in the morning. Her physical weakness is extreme and she is unable to walk more than a short distance without becoming very fatigued. Not infrequently she has to lie down for more than half of the day on account of this weakness. She has never fainted and does not seem to have had any attacks in which she was completely prostrated. The menstruation has been normal and she has not lost weight. She has not had any digestive disturbance and is positive that there has not been any diarrhea, but rather the opposite. The systolic pressure was 85 to 90 and the diastolic 55 to 60. The skin showed a very definite "white line." The blood count was normal. It is important to note that there was no evidence of pigmentation.

In such a case it is evident that different views might be held. One might regard the nervous disturbance as the essential matter, while another might say adrenal insufficiency. Naturally, the question may be asked, Does this represent an early stage of Addison's disease? If allowed to go on might this disease in its fully developed form have resulted? No one can answer positively, but I am inclined to say No to both questions. The results suggest a functional disturbance rather than organic change. The result of therapy is of interest. She could not rest entirely, so that she was advised to obtain as much rest as possible and given epinephrin by mouth, beginning with 5 drops of a 1 : 1000 solution after meals, gradually increased to 10 drops. The result was prompt and remarkable. She improved steadily and three months later her blood-pressure was 120 systolic and 90 diastolic. Her symptoms had gone and she regarded herself as

well. Now, after an interval of over a year, she has continued well and considers herself as perfectly healthy. Such a result is suggestive of some internal secretion disturbance which was readily corrected.

*Menopause.*—Disturbances of blood-pressure are common at this period, but greater attention has been paid to increased than to decreased pressure, yet in a certain number of cases the pressure is reduced. There is nearly always much nervous disturbance and one is usually in doubt as to which is the primary condition. There are many features suggestive of disordered function of the sympathetic system. This patient illustrates some of the problems:

She is forty six years of age and complains of nervousness, headache, and weakness. Her past history shows nothing of importance. She has never regarded herself as nervous and states that she has always done a good deal of hard work. For a year past she has been nervous, easily upset by trifles, and depressed at times. She has had headache, usually every day, and at times some dizziness. The periods have been irregular and she has had two in the last five months. She complains somewhat of what are described as "hot flashes or flushes." Examination shows that she is well nourished and has a good color. There is no exophthalmos and the thyroid gland is not enlarged. The thorax and abdomen seem normal. The deep reflexes are increased. The blood-pressure is 95 systolic and 55 diastolic. Investigation into the condition of the sympathetic system does not give results which enable us to state that she shows vagotonia or sympathicotonia; she shows some features of each. There is nothing to suggest myocarditis or arteriosclerosis. The Wassermann reaction is negative.

We are chiefly interested in the low blood-pressure, and it is interesting to speculate as to the cause. Naturally, some disturbance of internal secretion is suspected on the theory that one set of glands is going out of commission and the remaining active ones have not adapted themselves to the change. It is certainly worth a trial to determine whether internal secretion therapy will improve conditions. In such cases we may use the whole

substance of the ovary or the corpus luteum extract or a combination of several glands. In this case we have given her the corpus luteum extract, and if not successful, the others can be tried subsequently. As a result of this therapy the patient shows considerable improvement in her general symptoms, but her blood-pressure has not returned to normal, although it has risen to 110 systolic and 75 diastolic. She feels so much better that she insists on going home. (It is impossible to report the final result. She has not returned and has moved from her given address, so that she cannot be traced.)

**4. Nervous Disturbance.**—I do not desire to go into the question as to classification and the best terms to apply to particular forms of functional and nervous trouble. We probably all understand perfectly well what is meant when we describe a patient as being "tired out nervously." In this group there is no uniformity as regards blood-pressure, but in a considerable number of them hypotension is a marked feature. One is often puzzled to know how great a part this plays in the causation of the symptoms. Many of them have evident disturbance of the internal secretions, and it would be perfectly possible for one man to consider that this was primary and the nervous disturbance secondary, and for another man to take exactly the opposite view. My own feeling is that either disturbance may be primary. After a time a vicious circle is probably set up, the nervous disturbance upsets the internal secretions, and the internal secretions return the compliment. The following case illustrates some of the features of this form:

The patient is a young woman, aged twenty-four, who has been doing office work. She comes complaining of a great many symptoms, but lays most emphasis on the fact that she becomes so completely fatigued by 2 or 3 o'clock in the afternoon that it is difficult for her to keep at work through the remainder of the day. In addition, she has a good deal of headache, is very emotional, and at times quite depressed.

*Past History.*—There is no history of any acute infection. Some years ago she had headache, which was apparently relieved by glasses. Her digestion has been normal and there is no

history of any cough or shortness of breath. She has suffered somewhat from constipation. For the past three years her periods have been very scanty, but have always been regular. We find that she has gone through a good deal of nervous strain in the last two years. Some family trouble has contributed to this, the details of which need not be gone into.

*Present Illness.*—The exact duration of her symptoms is difficult to state; it probably goes back about three years. As she tells the history herself she first noticed some digestive discomfort which was never very serious, but troubled her occasionally. Then constipation became rather marked and has troubled her ever since. Later headache began, and this has continued until the present. She now has fairly severe headache nearly every day, which often tends to come on in the afternoon when she is particularly fatigued. Any little excitement or slight extra exertion brings on the headache rather acutely. She has kept at work, although this often required a good deal of special effort and forcing. Recently she has realized that she was easily upset and emotional, that tears came readily, and that she has periods of depression which seem to come without any evident cause. She has lost a certain amount of weight in the last three or four months.

*Examination.*—You observe that the patient's general physical condition seems fairly good. Examination shows that she is ordinarily well nourished, without anemia, and with no evidence of any mouth or throat infection. Naturally, in such a case we examine with particular care for evidence of thyroid disease and pulmonary tuberculosis. Physical examination of the chest shows nothing abnormal in the lungs and this is borne out by an *x*-ray examination. We cannot make out any enlargement of the thyroid and she has no signs which suggest hyperthyroidism. The epinephrin test for this is negative. The examination of the heart shows nothing abnormal except that the rate seems to vary rather easily. The sounds are clear, but the systolic pressure varies from 95 to 100 and her diastolic pressure is 60.

*Remarks.*—I think you can see that it is perfectly possible

to interpret her condition in different ways. One would expect that an individual with a blood-pressure below 100 would not be able to go through the day doing fairly hard work, as this patient has done, without tiring readily. On the other hand, we know that fatigue is a manifestation of certain forms of functional nervous disturbance. There does not seem to be any one definite test by which we can decide which is the primary condition. My own feeling is that in this patient the nervous upset, call it what you will, is probably the primary affair.

Subsequently the patient's earlier history was gone into more thoroughly, and it was discovered that she had been a rather shy, abnormal child, and had picked up some very curious misapprehensions with regard to her environment. She imagined that her family did not care for her, and for a period of her childhood was firmly convinced that her death would be a comfort to them. As she grew older she adjusted herself somewhat to her surroundings, but it is evident that she has never been able to do this completely. One of the objects of our treatment has been to try and help her to adjust herself to the conditions of life. We have kept her quietly in bed with hydrotherapy and general tonic treatment. She seems decidedly better in every way. (Three months later she is greatly improved as regards her nervous condition and the pressure is 120 and 90. This resulted from general measures and was not aided by any glandular therapy, suggesting that the disturbance in the nervous system was the principal cause.)

As we have discussed examples of some of the conditions causing low blood-pressure, I now bring before you a patient for diagnosis as to the cause: The patient is a white male, aged forty-five, a stoker on an Atlantic steamer, who comes complaining of dizziness.

*Past History.*—He had measles in childhood and typhoid fever at the age of twenty-five, but no other acute infection. He has spent many years in the British Army and went to France in 1914 with the early contingents. He was wounded by shrapnel in several places and as a result of one wound lost his right eye. He had also been a soldier in the South African War and



had a slight injury to the head by a splinter of shell. He has not used alcohol to any extent and there is no history of any venereal disease. His wife has not had any miscarriages and has 4 healthy children.

*Present Illness.*—He had been perfectly well previously, and on November 10, 1919 began to vomit without any evident cause. He also felt very dizzy, this being so severe that he had to grasp objects to keep from falling. He continued at work until November 12th, when he collapsed and had to stop working. Since that time he has improved and at present feels dizzy only once in two to three hours. He has not vomited since the onset and has had no other symptoms.

He is perfectly positive that before the onset he had been absolutely well and had worked hard without any fatigue and without the slightest shortness of breath. He lays particular stress on the dizziness, and states that whenever he was lying down he has felt perfectly comfortable.

*Examination.*—The patient is fairly well nourished, healthy looking, perhaps a little below weight, and has a good color. There is no dyspnea or cyanosis, the tongue is clean, the thyroid is not enlarged, and examination of the thorax shows nothing of any importance in the heart or lungs. There is no shock or thrill felt over the heart, no increase in dulness, and the heart sounds are clear throughout and of good quality. The pulse is regular, rather soft, and with an average rate of 64. The abdomen is negative. The reflexes are increased. The systolic pressure varies from 95 to 110, the diastolic is about 70. The blood count shows nothing abnormal and the Wassermann reaction is negative. The urine is normal in every way. The temperature record is a little below normal, generally being between 97° and 98° F.

DR. MCCRAE: The most striking abnormal finding in this patient is the low blood-pressure. His reflexes are rather exaggerated and you see that he shows fairly marked dermatographia. Of particular importance, however, is the fact that he does not show the typical "white line." Mr. A., what do you regard as the probable explanation for the hypotension in this patient?

STUDENT: I should say that he probably has myocarditis.

DR. MCCRAE: What reasons have you for making this diagnosis of the cause of the low pressure?

STUDENT: There is nothing much more than the low pressure itself. Certain of the conditions which have been mentioned can be excluded, such as an acute infection and any form of shock.

DR. MCCRAE: If there is no other evidence in favor of myocarditis, let us see what points we can bring against it. What would you say is the most frequent and very often the most common early symptom of myocarditis?

STUDENT: Shortness of breath.

DR. MCCRAE: That is correct and a point well worth keeping in mind. Can you add anything to your answer to make it a little more complete?

STUDENT: The fact that the shortness of breath comes on particularly with exertion.

DR. MCCRAE: There is no doubt of that, not meaning, however, that every patient with shortness of breath on exertion necessarily has myocarditis. Its occurrence, however, is always an indication that we have to exclude myocarditis. Now let us go back a little in this man's history. He has been doing extremely heavy work as a stoker on an Atlantic steamer which would probably cause shortness of breath in most of us if we had to take hold of it today. He is perfectly positive that he had been able to do his work without any difficulty and that he has never had any shortness of breath. Does this speak for myocardial insufficiency?

STUDENT: No, it does not.

DR. MCCRAE: Those of us who have examined the patient have not been able to discover any evidence of myocarditis. Naturally, this does not exclude some change in the heart muscle, but we must regard it as unlikely. Which of the other causes should we consider next?

STUDENT: Nervous disturbance.

DR. MCCRAE: This is probably the explanation. The patient went through some of the hardest days of the war and the regiment to which he belonged suffered very severe losses in the

fighting of 1914. He was severely wounded and had a slow convalescence. The findings are compatible with such a diagnosis. Some of you have seen patients in the wards who went through active service with no evident disturbance, but after discharge have showed evidence of trouble of various kinds, especially in the way of functional nervous conditions. The strain of active service took much more out of many of these men than they realized. Have we to think of anything else?

STUDENT: Disturbance of the function of the adrenal glands.

DR. MCCRAE: This has to be considered and may be associated with the other condition. It seems wise to give him epinephrin for a few days and watch the result.

The patient was shown again three weeks later. The use of epinephrin did not seem to have any marked influence on the blood-pressure. He was kept in bed, given full diet, and an occasional dose of a hypnotic at first, as he had some difficulty in sleeping. He improved steadily, gained markedly in weight, the nervous symptoms decreased, and on discharge after six weeks stated that he felt perfectly well and able to return to work. His pressure was at the low limits of normal. The final opinion was that the low pressure was secondary to his general nervous condition.

It may be of interest to consider the study of a series of 100 consecutive cases of low pressure, those due to acute infectious disease not being included. As in some of these there were two or more causal factors the classification is only approximate, and the headings represent the most marked condition.

1. **Nervous Disturbance.**—This took first place and was the cause in 38 cases. It is evident that internal disturbance undoubtedly played a part in some of these cases; perhaps some would say in all. In studying these patients my expectation was that a large number would prove to be due to internal secretion disturbance, and my hope was to find it in a larger number than proved to be the case. So far as the observations went there was no striking evidence of such disturbance in the cases of this group.

2. **Myocarditis.**—There were 25 cases which came under

this heading. It is worthy of note that in general the lowest figures were found in this group.

3. **Internal Secretion Disturbances.**—The number here was surprisingly small—only 8. I quite realize that some would shift many from the first group to this one. Of these cases, 3 showed Graves' disease and 2 myxedema. The others showed polyglandular disturbances.

4. **Anemia.**—This was the cause in 3 cases, which seems a small number. Of course some of those in the other groups showed some degree of anemia, but it did not seem to play the most important part.

5. **Miscellaneous.**—The remaining cases came under various heads, which in general represent some form of chronic disease—nephritis, syphilis, arthritis, epilepsy, etc.

**Diastolic Pressure.**—You may ask how important this is and what relationship it bears to the systolic pressure. I do not feel that any definite answer can be given. The lowest average diastolic pressures were found in the group with internal secretion disturbances. The highest diastolic pressures were found in the group with nephritis; in some of these the difference between the two pressures was small. The same was true of some of the cardiac cases, in one of which the difference was only 10 mm. Hg. In an occasional case it is not possible to estimate the diastolic pressure for the reason that the sounds are not heard. I have no explanation to offer for this.

**Symptoms.**—In a condition which is not a disease it is not likely that there will be clear-cut symptoms, yet hypotension gives some which are suggestive. The most striking are weakness, headache, dizziness, and fatigue. The first and last offer no special peculiarity except that they are particularly brought on by exertion which is continued for any time. The headache is often characteristic in that it is often due to a change in position, as when the patient rises from a lying to a sitting position or bends over. It may last for a few minutes only or persist for an hour or two. The probable explanation is that the circulation does not adapt itself quickly to the change from the horizontal to the erect posture. With it there may be dizziness which in some patients is severe.

**Diagnosis.**—The recognition of the existence of low blood-pressure is easy, but we must try and find the cause back of it. In this it is well to go systematically through the possibilities. Myocarditis and anemia are usually easily recognized. The pressure of nervous disturbance does not give much difficulty, but its importance may not be easy to determine. Probably the greatest difficulty comes in deciding how much part internal secretion disturbance plays. In a small group there seems no question of adrenal insufficiency as shown by the marked weakness and the presence of the "white line." The therapeutic test of giving epinephrin is of value in some instances; a prompt response is suggestive.

**Treatment.**—Evidently this will vary with the cause, which must be determined before intelligent therapy can be carried out. In the cases of acute infectious disease prevention is important, especially from measures directed to elimination and those which support the circulation. Hydrotherapy—inside and out—is one of our greatest aids. Among the measures directed to the circulation itself digitalis should be considered and its use before the symptoms are marked is important. From internal secretion therapy in this group I have never obtained definite results. Some report benefit from the giving of epinephrin in typhoid fever. From the use of pituitary extract there have been very fleeting effects, but there does not seem to be any permanent result.

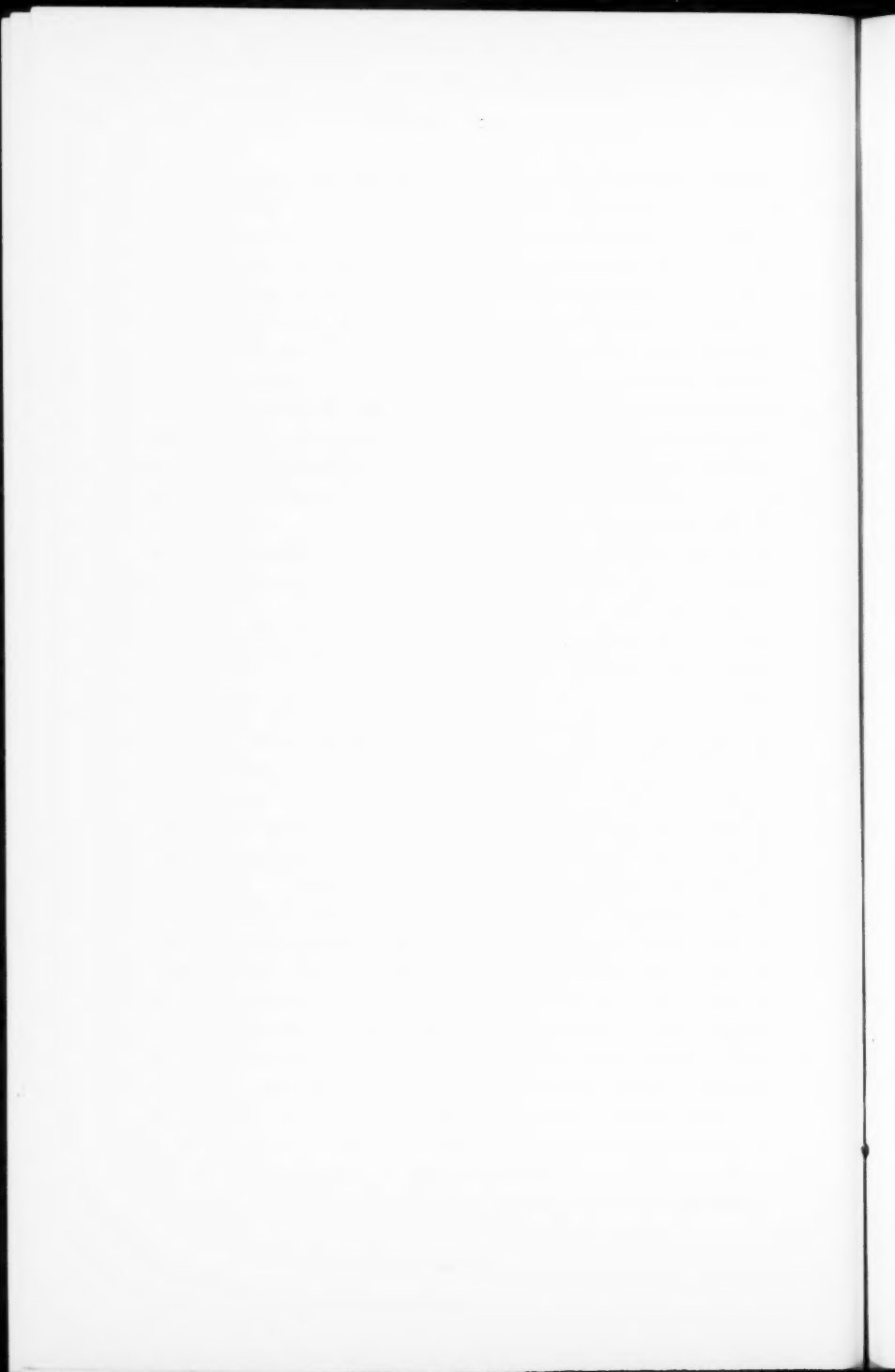
In the group in which *nervous disturbance* is prominent the usual measures for such conditions should be carried out with especial emphasis on rest. It is a question to what extent we should adopt measures directed specially to the low pressure. If the patient is in bed, hydrotherapy is indicated and massage may be added with advantage. If the patient is up and about, a certain amount of rest is advisable, with which gentle exercise may be given. It is an advantage if the exercises are done with the patient lying flat. As many of these patients are of the thin, visceroptotic type, an abdominal support is of value. A careful study should be made to decide if there is any evidence of internal secretion disturbance. If there is proof of this or

sufficient suspicion to justify glandular therapy, one has the difficulty of deciding which extract is best. In this there is often difficulty, and there does not seem to be any set rules that we can use. It is usually safe to give epinephrin by mouth in doses of 5 drops of the 1:1000 solution three times a day. This dosage may be gradually increased to double the amount. In some patients the use of a mixture of gland extracts seems more useful—the dose of each should be small. Ergot is sometimes of great use in this group and in some cases has a marked effect in aiding the headaches which are so common. It may be given in dram doses of the fluidextract or in corresponding doses of some of the special preparations. It sometimes gives relief to the headache in the very nervous patients.

In the cases in which there is reasonable evidence of adrenal insufficiency, the epinephrin should be given in the dosage mentioned, but this may be increased even to larger amounts. Various combinations with other glandular extracts may be tried, but it is not easy to state the indications for any particular one.

In women at the *menopause* the administration of ovarian or corpus luteum extract is indicated. Some do better with one and some with the other. A combination of many glands sometimes does better. In all the cases in which a nervous element is evident special care should be given to the general method of life and to the effort to remove disturbances of all kinds. Rest and exercise should be very carefully prescribed.

In **conclusion** let me emphasize again the fact that low blood-pressure is a symptom and not a disease. Its presence suggests the need for careful study to determine the underlying cause. It is on this that proper therapy has to be based. Let me suggest that each of you can make studies which would be of great value in this question of low pressure, especially in the group associated with internal secretion disturbance. You will be able to study your patients over long periods of time, which is difficult to do in a hospital practice. A period of long observations gives you also the opportunity of a careful study of the comparative value of various forms of treatment.





## CLINIC OF DR. ELMER H. FUNK

JEFFERSON HOSPITAL

---

### MALIGNANT DISEASE OF THE LUNG

**Malignant Disease Often Mistaken for Tuberculosis; Statistics Relating to Primary and Secondary Growths. Five Case Reports. Discussion of Etiology and Pathology. The Symptoms and Signs: Shortness of Breath, Cough, Pain, Pressure Effects, General Symptoms. Charts Illustrating Five Groups. Pathologic Specimens. References.**

ALTHOUGH new growths in the lung occur relatively infrequently, their recognition is often possible if due care is exerted in the study of the patient. They are, for the most part, mistaken for pulmonary tuberculosis, and the expense of sanatorium care may cause a hardship, especially among those of limited means. The presence of such a patient in a tuberculosis sanatorium fills a bed which may be used more advantageously for a tuberculous patient, and, moreover, such a patient with the inevitably fatal course discourages many fellow patients. Among 1200 patients sent into the wards of the Chest Department of the Jefferson Hospital with the diagnosis of advanced tuberculosis, 72, or 6 per cent., were incorrectly diagnosed, and of this number 5 were instances of malignant disease of the lung. Stated in another way, 1 patient among every 250 referred to us with the diagnosis of advanced pulmonary tuberculosis showed malignant disease instead, and among those incorrectly diagnosed, malignancy was present in 1 out of every 15.

Ash collected autopsy statistics from a number of tuberculosis institutions, and found among a total of 551 autopsies that 61 were non-tuberculous (11 per cent.). Among these there were 7 instances of neoplasm.

Furthermore, the possible value of operative interference in the event of a correct diagnosis must be kept in mind. The experience and investigations of Seydel, Guyot and Parcelier, Garre, Kuttner, Lenhartz, and others point to such a possibility. Seydel made a careful study of 55 reported cases with regard to this point, and concluded that operation might have been considered in 4 and was actually performed in a fifth, with satisfactory results. Guyot and Pareclier showed that operative interference in pulmonary sarcoma (which apparently is more amenable to operation than carcinoma) might have been considered among 17 of the 56 reported cases which they studied. Before presenting our case records a word may be said with regard to the frequency of malignant disease of the lung.

The statistics of Passler show that among 1000 cases of malignant disease there were 16 cases of primary carcinoma and 5 cases of primary sarcoma of the lung. Seydel found 184 tumors of the lung and pleura among 10,829 autopsies; and of these, 16.8 per cent. were primary and 83.2 per cent. secondary.

Adler in 1912 was able to collect for his monograph on primary malignant growths of the lungs and bronchi 374 cases of primary carcinoma and 90 cases of primary sarcoma of the lung. He calls attention to the fact that the increase in the percentage of lung tumors among the more recent autopsy records is due to the increased attention paid to these types of tumor and the greater care and more extensive microscopic investigation with which autopsies are carried out at present. Stokes, in his text-book on Diseases of the Chest, called attention in 1837 to the fact that in his experience lung tumors were by no means so rare as was generally assumed.

Secondary carcinoma may arise from an initial focus in the abdomen or from extension of a mammary, esophageal, or thyroid tumor. Secondary sarcoma most often originates from a primary growth in one of the long bones. There are two types of malignant tumor allied to carcinoma and sarcoma which appear as secondary growths in the lung, namely, hypernephroma and malignant deciduoma. Woolley collected 22 cases of the hypernephroma, of which 13 showed metastasis in the lungs, and

Stevens states that of the cases of malignant deciduoma fully 50 per cent. show pulmonary involvement.

#### CASE REPORTS

**CASE I.—Primary Malignant Disease of Left Lung (Massive Carcinoma). Referred with Diagnosis of Pulmonary Tuberculosis. Extensive Lung Involvement with Sputum Negative for Tubercle Bacilli.**—L. H., aged forty-seven years, male, white; presser by occupation.

*Family and personal history negative.*

*Present Illness.*—There has been a gradual loss in strength and weight during the past two years. One year ago he was compelled to stop work because of weakness. During this time there developed cough and expectoration, and three months ago hoarseness. There has been a gradual and increasing shortness of breath, which during the last few months was present constantly, even while at rest. He has a constant feeling of pressure and of aching on the left side, but no severe pain. His best weight was 180 pounds two years ago, and present weight 111 pounds.

*Physical Examination.*—Patient was generally emaciated and pallid. The fingers showed marked clubbing. The chest examination revealed marked flattening on the left side, with a compensatory bulging of the right side. Entire absence of expansion on the left side. The percussion note was flat anteriorly and posteriorly from apex to base on the left side. There was a suggestive Grocco's sign on the right side. The right side was generally hyperresonant. Breath sounds over entire left side were distant and scarcely audible; vocal fremitus and vocal resonance were absent. In the left axilla there was a small gland about  $\frac{3}{4}$  inch in diameter, firm, movable, and painless. There was a small nodule in the skin on the inner side of the left arm. The chest wall on the left side was firm and the interspaces felt as though they were infiltrated with the tumor material.

The *x-ray examination* by Dr. Manges revealed the following: The left side of the chest was dense throughout. No shadows were visible except in the apex, where the rib shadows showed

faintly. The heart was displaced to the right and there was pleural adhesion to the diaphragm on the right side. There was infiltration about the hilus of the right lung extending out to the third interspace. The x-ray diagnosis was malignant disease, probably carcinoma of lung.

Repeated sputum examinations were negative for tubercle bacilli. The Wassermann test was negative. The blood examination revealed a moderate secondary anemia with 10,500 leukocytes. The temperature charts of two admissions show nothing abnormal except an occasional elevation in the afternoon temperature to 99.0° F. or a fraction above.

**CASE II.—Secondary Malignant Disease (Carcinoma) of Left Lung (Primary Growth in Stomach). Referred with Diagnosis of Pulmonary Tuberculosis with Gastric Symptoms. Sputum Negative for Tubercle Bacilli.**—A. T., aged forty years; male, white; expressman by occupation.

*Family and personal history* negative.

*Present illness* began one year ago with stomach symptoms, which he stated consisted principally of pain in the stomach which gradually became worse and "went into the chest." He had a moderate cough which was productive of a moderate amount of sputum which three times during the past year contained considerable blood. He has become short of breath on exertion during the past few months and recently developed slight hoarseness which has persisted.

*Physical examination* revealed an emaciated, pallid adult. Cervical lymph-glands enlarged and firm. Fingers showed distinct clubbing. Chest examination showed general limitation of expansion, especially marked in the left upper chest. Flatness on left side from apex to fifth rib anteriorly and to angle of scapula posteriorly. Breath sounds tubular over the same area and associated with numerous fine crackling râles throughout the entire left lung. The right lung was clear throughout. The abdomen is normal in contour. Liver outline normal. Spleen not palpable. No masses felt.

Sputum examinations were repeatedly negative for tubercle

bacilli. The Wassermann test was negative. Blood examinations showed moderate anemia with 8200 leukocytes. The temperature chart is herewith reproduced (Fig. 222). The x-ray of

## JEFFERSON HOSPITAL

Name Alexander Suber Age 48 Date Sept 14 1916 Register No. 9289

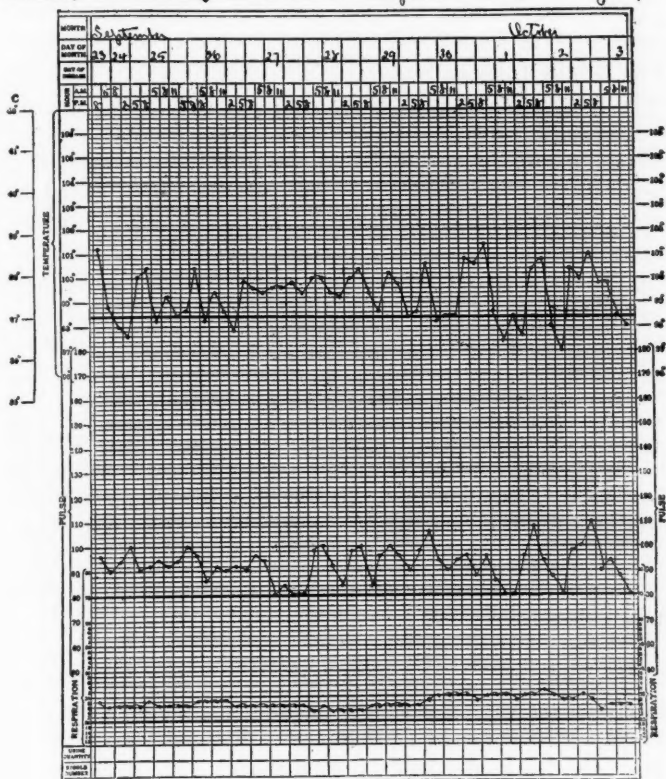


Fig. 222.

the chest reveals a mass in the left lung projecting from the mediastinum into the lower portion of the left upper lobe. It suggests a gumma or new growth—probably the latter. The x-ray of the abdomen reveals deformity at the pylorus, but not

especially distinctive of carcinoma. Dr. Reh fuss, in reporting his gastro-intestinal examination, stated that he found considerable pus and bacteria in stomach residuum. He thinks it is most likely swallowed pus coming from the lung. It may be, however, that the process in lung is secondary to gastric lesion. The gastric findings were not conclusive. The second x-ray report was as follows: "The infiltrated mass in the left upper lobe is fully twice as large as on the former examination. We believe that it is not tuberculous—probably carcinoma."

*Comment.*—It must remain an unsettled question, unless we secure an autopsy on this patient, as to whether the lung tumor is primary or secondary. The fact that the gastric symptoms antedated by a considerable period the development of respiratory symptoms suggests that the pulmonary lesion is secondary. On the other hand, the inconclusive gastric studies would seem to indicate that the gastric symptoms are perhaps secondary to the lung lesion. Our feeling, however, from the clinical study of the patient, is that the lung lesion is the secondary lesion.

**CASE III.—Secondary Carcinoma of Lung (Primary Growth in Esophagus). Referred with Diagnosis of Tuberculosis with Laryngeal Ulceration and Dysphagia. Sputum Repeatedly Negative for Tubercle Bacilli. Wassermann Negative.**—I. R., aged sixty-three years; male, white; rag dealer.

*Family History.*—Mother died at fifty-seven years of age of tuberculous pneumonia.

*Personal history* negative.

*Present illness* began six months ago, when, following an injury to the chest incident to falling down stairs, he began to cough and lose weight and strength. The pain of the injury gradually disappeared and was subsequently followed by hoarseness and pain in the throat and difficulty in swallowing. During the past two months difficulty in swallowing has become quite marked and he is now able to ingest only liquids and soft foods. Two weeks ago he noticed swelling of his ankles. His best weight was 237 pounds some years ago and his present weight is 97 pounds.

*Physical examination* revealed an emaciated, cachectic male, with prominently enlarged, firm cervical glands. Slight clubbing of the fingers. The larynx is markedly congested, but no ulcera-

## JEFFERSON HOSPITAL

Name Space Kuller Age 63 Date Sept 15 1916 Register No. 42510

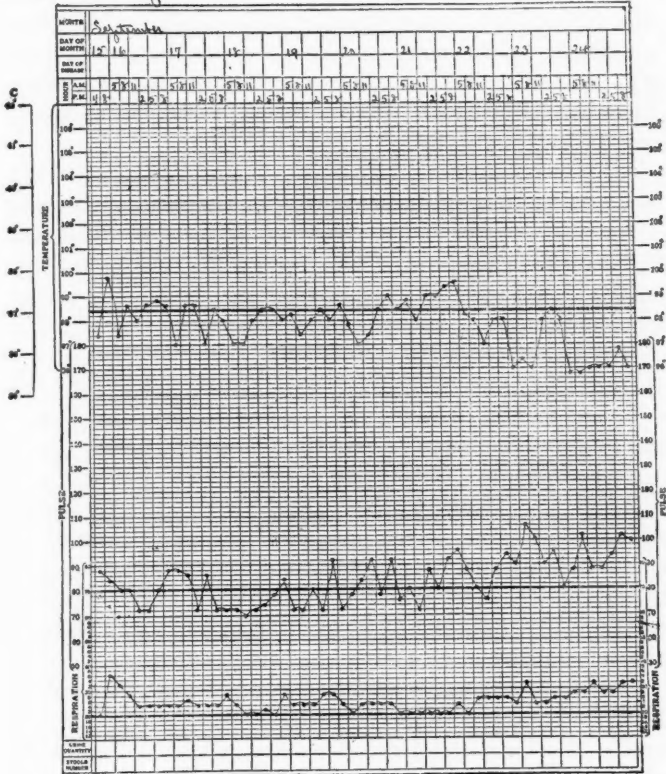


Fig. 223.

tion is seen. Chest examination revealed marked emaciation. Breath sounds were generally harsh and of a tubular quality, with scattered crackling râles throughout the entire pulmonary



area, especially at the apices. Patient was referred to the hospital with a diagnosis of pulmonary tuberculosis associated with stricture of the esophagus, probably malignant. The sputum was repeatedly negative for tubercle bacilli. The Wassermann test was negative. The blood showed moderate anemia with 11,400 leukocytes. Temperature chart is herewith reproduced (Fig. 223). The x-ray examination showed the following: There is a stricture of the esophagus about 3 inches above the cardiac orifice, which is probably malignant. The lung structure is studied with fairly circumscribed shadows, most likely due to metastatic new growths.

**CASE IV.—Sarcomatosis Involving Pleuræ, Multiple Growths in Various Organs. Lungs Showing at Autopsy Subpleural Nodules, Congestion, and Edema. Sputum Negative for Tubercle Bacilli.**—J. D., aged forty-eight years; male, white; boilermaker by occupation.

*Family and personal history* negative.

*Present Illness.*—Following "rheumatism" (swollen joints) of several months' duration he developed cough, expectoration, and loss in strength and weight, which have continued up to the present time. No hemoptysis. Dyspnea was present, even while at rest. Hoarseness has existed for the past two weeks, during which time he has also developed night-sweats. He stated that his urine was at times dark, and we assumed, from his description, that it may have contained blood. His best weight was 190 pounds in 1900. In 1906 he weighed 142 pounds, and on admission in 1914 he weighed  $122\frac{3}{4}$  pounds.

*Physical Examination.*—Patient is undernourished and pallid. Dyspnea is present. Lips slightly cyanosed. Chest, respiration rapid—40 per minute. Expansion fairly equal. No impairment of percussion note. Auscultation reveals numerous râles of all types, especially in lower portions of chest, where the crepitations are superficial and suggest pleural origin. The heart shows marked weakness of first sound, but no murmurs audible. The abdomen is negative. Slight pretibial edema.

The sputum examinations were repeatedly negative for



tubercle bacilli, although many streptococci, staphylococci, and other unidentified organisms were found. It was frothy, yellowish brown, and mucopurulent. The urine contained a decided trace of albumin with many granular casts. The blood examination revealed 31,000 leukocytes. The Wassermann test was negative. The temperature record reveals an intermittent pyrexia not unlike that of Case II (Fig. 222). Autopsy revealed the following: Multiple sarcoma of pleura (see Fig. 229), liver, peritoneum, vertebræ, and mediastinal and retroperitoneal lymph-nodes, congestion and edema of lungs, chronic adhesive pericarditis, chronic diffuse nephritis, chronic adhesive peritonitis, beginning atheroma of aorta, chronic gastritis, fatty degeneration of myocardium, edema of brain, congestion of spleen, nephrolithiasis.

**CASE V.—Secondary Sarcoma of Left Lung—Extension from Chest Wall.** Referred with Diagnosis of Pulmonary Tuberculosis with Tuberculous Abscess of Chest Wall. Sputum Negative for Tubercle Bacilli.—P. H., aged forty-eight years; male, white; clothing operator.

*Family history* negative.

*Present illness* began six months ago with pain in left side which has persisted up until the present time. Six weeks ago the patient noticed an enlargement in the region of the left scapula. Recently patient has noticed shortness of breath upon exertion. He has a slight cough, which is now productive. No hoarseness. He has been feeling quite weak for some time.

*Physical examination* revealed on inspection the following: Posteriorly on left side, extending from above spine of scapula to about 2 inches below angle and between spinal column and under surface of scapula, was a mass about 3 inches in diameter and slightly elevated above the surrounding surface; firm, solid, and smooth; at points softer than others and firmly fixed to chest wall. Slightly tender to touch. Skin slightly adherent. The chest wall extending beyond the tumor proper felt unusually firm and indurated. The heart was distinctly displaced toward

the right side. The temperature chart is herewith presented (Fig. 224).

## JEFFERSON HOSPITAL

Name Philip Weiss Age 48 Date 9-12-1914 Register No. 81616

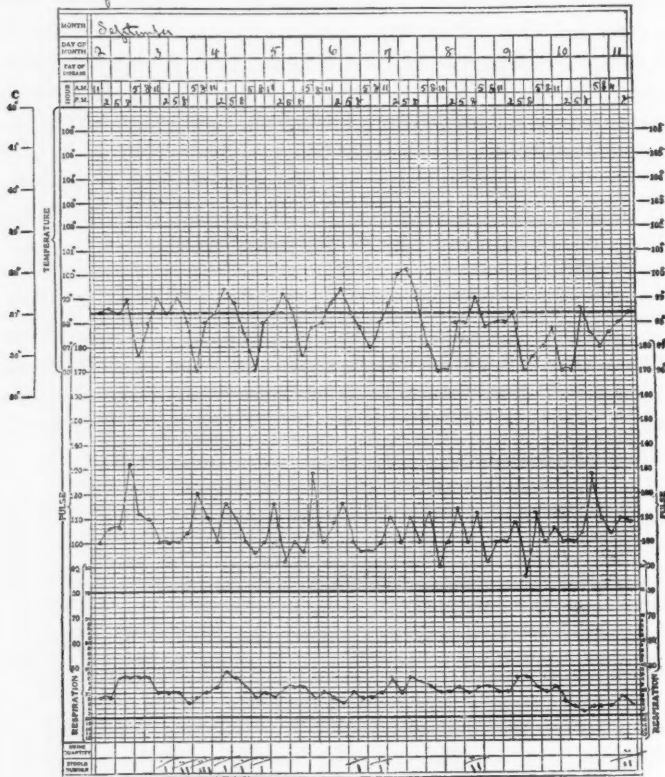


Fig. 224.

*Clinical Note.*—After admission to the hospital the dyspnea became quite marked and continued until death. Pain was alleviated only by opiates. The left chest was aspirated and 1000 c.c. of thin, bloody fluid removed. The removal of the fluid caused no change in patient's condition. The examination of

the fluid revealed a large number of red cells and cellular débris. Many degenerated cells staining poorly were found. Spreads and cultures for bacteria were negative. Inoculation into guinea-pigs was negative.

The *x-ray examination* revealed the following: Very marked density of the upper middle left lung, which appears to be consolidation rather than pleural thickening; the lower latter portion of the lung is emphysematous, the upper right portion shows two rather small, very dense, circumscribed areas. A portion of the fifth rib posteriorly has disappeared and the sixth shows distinct involvement. The lesion is most likely sarcomatous, with involvement of the ribs and chest wall.

The sputum examination was negative. The blood showed a moderate anemia with 12,000 leukocytes. The urine was negative.

#### ETIOLOGY AND PATHOLOGY

There is as yet no known primary cause of malignant growths, and yet Ewing, in his recent work on the neoplastic diseases, states, with regard to primary carcinoma of the lungs, that "the chief etiologic factor is tuberculosis." He quotes Schwalbe, Friedländer, and Peronne, who found squamous-cell carcinoma developing in the walls of a tuberculous cavity. Schwalbe was able to demonstrate the coexistence of tuberculosis in 3 of the 10 cases which he autopsied. Wolf believes that the coexistence of the two diseases is common. He found 23 instances of tuberculosis in 31 of malignant disease; on the other hand, Fowler, in another series of 30 cases, found only 2. In the writer's opinion it is questionable whether tuberculosis plays any part in the predisposition to malignant disease of the lungs. Certainly it is not more than a very minor part. Considering the great frequency of pulmonary tuberculosis, latent and active, the infrequency of pulmonary malignant disease, it is difficult to imagine a relationship other than coincidental. On the other hand, we are not able to accept Rokitansky's dictum that carcinoma and tuberculosis are incompatible diseases. We do feel, however, that the association is largely coincidental. Crazet, in his Paris Theses of 1878, expressed it well when he said, "The cancerous easily become tuberculous, but the tuberculous do not easily

become subject to cancer." When the two diseases are associated the problem of the dual diagnosis is a difficult one. The presence of the tubercle bacillus in the sputum may establish the one. The x-ray may help in the differentiation of the other. In our experience the cases where the two diseases are associated have been exceedingly few.

Although trauma has for a long time been considered a potent factor in the etiology of the malignant disease, the reported cases showed singularly few instances in which gross traumatism was present. Aufrecht reports several instances in which it would appear that trauma played a part. A fall from a ladder, a falling beam, a blow from a piece of iron, a kick have been noted as antecedents of the malignant disease and appeared to bear a definite casual relationship. Smaller irritations, such as dust, etc., have also been supposed to play a part in the etiology and may explain the predominance of males over females affected. Landis, who has so thoroughly investigated the various phases of the dust occupations, tells me that he has been unable to establish any such relationship. He has had an unusually large experience among potters and he has not seen a single case among them.

The relationship of age and of sex has been worked out by Adler, who found among 374 cases of carcinoma of the lungs 269 males, or 71.9 per cent.; 93 females, or 24.8 per cent.; 12 in which the sex was not stated. In the same way, among 94 sarcoma cases, 63, or 67 per cent., were males; 28, or 29.7 per cent., females; 3 where sex was not stated. The age distribution was found to be as follows:

Carcinoma.		Sarcoma.	
Age not stated. . . . .	18	Age not stated. . . . .	9
1-10. . . . .	0	1-10. . . . .	6
10-20. . . . .	6	10-20. . . . .	12
20-30. . . . .	10	20-30. . . . .	14
30-40. . . . .	30	30-40. . . . .	19
40-50. . . . .	78	40-50. . . . .	14
50-60. . . . .	113	50-60. . . . .	12
60-70. . . . .	94	60-70. . . . .	3
70-80. . . . .	23	70-80. . . . .	5
80-90. . . . .	2		
	374		94

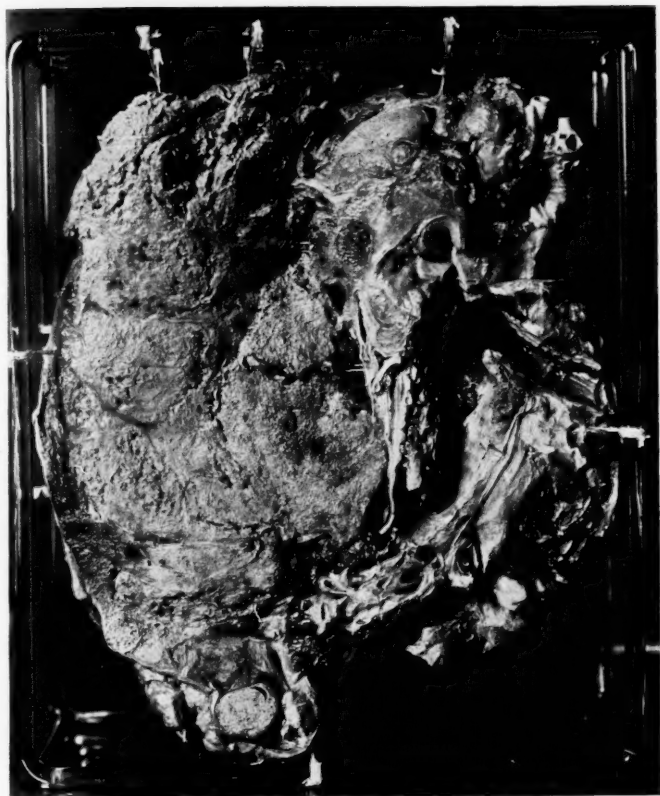


Fig. 225.—Specimen showing *primary carcinoma of right lung*. Note that the upper three-fourths of lung is involved in a lobulated growth which at autopsy was extremely soft and friable. The lower portion contains several isolated tumor nodules. The external surface, which is not visible, shows a thickened pleura. There were metastases to the left lung, liver, mediastinal and retroperitoneal lymph-nodes. The subject was a white male, twenty-six years of age, who during life was diagnosed as having chronic pulmonary tuberculosis. (From the Jefferson College Museum.)

It is not within the province of this clinic to discuss the pathology of pulmonary malignant disease, except in so far as it allows of a better understanding of the clinical manifestations.



Fig. 226.—Specimen showing *metastatic carcinoma of left lung*. Note the circumscribed nodule in the upper portion of the lung which has been cut in two in the section. Just below are seen the cut surfaces of the carcinomatous lymph-nodes. The external surface of the lung, which is not in view, showed several small nodules just below the pleura. The specimen was secured at autopsy on a white female aged twenty-seven years, who three months prior to death had noticed pain in the breasts and middle of back, with loss of strength and weight. On admission to the hospital the patient presented an emaciated condition, with pronounced weakness. Physical examination revealed slight enlargement of the thyroid, palpable cervical glands, a hard swelling in both breasts about the size of a walnut, adherent

We are at once confronted by the fact that a considerable variation in the gross pathology is entirely in keeping with the considerable variation in the symptoms and signs which we find clinically. An examination of some gross specimens is fundamentally necessary if we are to understand the variations in the clinical manifestations. I wish therefore to present for your inspection some specimens from our museum at the Jefferson College through the courtesy of Professor Coplin (Figs. 225-229).

### THE SYMPTOMS AND SIGNS

The symptoms may be latent, and this is particularly true in late metastases to the lungs when the growths are few and small. Small deep-seated primary neoplasms may give no evidence of their presence. We have found this to be true in several patients dying from other causes. In most instances, however, and especially in primary growths, very definite symptoms and signs are present. These vary, dependent on the point of origin, location, size and rate of growth. The local symptoms are dyspnea, cough, pain, and pressure effects. The general symptoms are those of malignant disease elsewhere in the body.

**1. Shortness of Breath.**—The most striking symptom is the constantly increasing shortness of breath. It occurs early in those patients in whom the neoplasm originates from or near the bronchus, with a corresponding interference with the ingress and egress of air; and in basal cases with early involvement of the diaphragm. It is present some time or other in every case of primary neoplasm of the lung, and, once established, it becomes

---

to the skin and possessing the usual characteristics of mammary cancer. The maxillary glands were enlarged and firm. Examination of the chest revealed impairment of resonance at both bases, with many fine crackling râles. The apices showed no special signs except perhaps prolonged expiration. The heart sounds were feeble. The clinical diagnosis of mammary carcinoma was made with metastasis to the lymph-nodes and perhaps to other organs, but the involvement of the lungs could not with certainty be made out. The signs in the lungs were more those of pulmonary congestion due to myocardial weakness. Autopsy confirmed the diagnosis and showed metastasis to the lung, liver, and vertebra. (From the Jefferson College Museum.)





Fig. 227.—Specimen showing *secondary hypernephroma of right lung*. Note that the neoplastic process involves mostly the upper portion of the lung—the site which is usually involved in tuberculosis. The primary growth was in the kidneys and metastatic growths were distributed in the right lung and pleura, supraclavicular and peribronchial lymph-nodes. The subject was a colored male aged thirty-five years, who was thought to be suffering from pulmonary tuberculosis. No record of the sputum examination can be obtained and the clinical notes of this patient, who died in another hospital, are exceedingly incomplete. (From the Jefferson College Museum)





Fig. 228.—Specimen showing *secondary hypernephroma of left lung*. Note the large, necrotic, encapsulated mass in lower lobe. A second smaller mass is seen directly above. The external surface, which is turned away from the observer, shows the subpleural aspect of the tumor with its smooth and rounded surface. The overlying pleura is hyperemic. The primary focus was found in the kidney and metastatic growths were present in the heart, lung, diaphragm, and spleen. A specimen was secured at autopsy on a male, black, aged thirty years. The clinical history was of an onset three months prior to death, with pain in the back and chest, slight pain in the abdomen, vomiting and constipation, productive cough, and profuse night-sweats. On admission to the hospital he was somnolent, very weak and emaciated, breath foul, enlarged cervical lymph-nodes. Chest examination revealed an increase in the respiratory rate, limited expansion, general hyperresonance, except in the left axilla, where the note was dull. Many râles over the entire chest. The abdomen was distended with fluid. A provisional diagnosis of tuberculosis was made, but revised before autopsy to hypernephroma, with metastases to the lung. (From the Jefferson College Museum.)



Fig. 229.—Specimen showing *multiple secondary sarcomata of lung and pleura*. The clinical history is described in the text (Case IV). Note the numerous small nodules on the pleural surface facing the observer. Several of the larger ones are indicated by the arrow points. On the opposite side of the specimen the cut surfaces of the lungs are noted to contain a few subpleural nodules. (From the Jefferson College Museum.)

increasingly more marked. It is more or less proportionate to the rate and size of growth. A slow-growing tumor may approach considerable size and produce less dyspnea than a considerably smaller tumor similarly located and of a rapidly grow-

ing nature. The writer recalls seeing a lung specimen from a patient who died from cancer of the prostate. A few weeks before death there was sudden onset of cough, dyspnea and cyanosis, and signs suggesting an intense bronchopneumonia. At autopsy the lungs were studded with small, pinhead size nodules resembling miliary tubercles, but which microscopic study revealed to be malignant metastases.

2. **Cough** is early and troublesome in those cases in which the location of the tumor is in close proximity with the bronchial tree. Situated near the tracheal bifurcation it may give rise to a distressing, hollow cough having a "brassy" sound not unlike that occurring in aneurysm. Stridulous breathing is common in this type of malignant disease. When the bronchial wall is involved the cough is frequently quite intense and associated with an abundant expectoration which is frequently blood tinged. There may be frank hemoptysis. It may be the first evidence of the disease. When the tumor originates in or near the pleura the cough is not unlike that of ordinary pleurisy, frequent, dry, and associated with pain in the side. In some instances where the tumor originates in the lung structure proper, cough may be absent until it has reached a considerable size. The cough, once established, is, like the shortness of breath, progressive, and little likely to become ameliorated except by the use of sedatives. The expectoration is not characteristic—it may be mucoid or mucopurulent, and in a few instances, where the tumor is undergoing central necrosis, it may be extremely profuse and fetid. It is often blood streaked. The so-called currant jelly sputum is only seldom found. When bronchiectasis is associated the cough may assume a paroxysmal type and be associated with a profuse fetid sputum.

3. **Pain** on the affected side occurs early in those instances in which the tumor originates in or near the pleura, when it is not unlike that of ordinary acute pleurisy. The acute pain may clear up, to be replaced by a more or less constant aching pain due to progressive infiltration of the chest wall, as in one of our patients. The pain may be absent or present only occasionally throughout the entire course of the disease in those tumors which

do not extend to the pleura. In growths originating from the mediastinum pain is a common symptom and may be the only one. It is well to recall, however, that in a number of cases its presence was exceptional, or occurred late in the disease. It is not within the purpose of this clinic to discuss mediastinal growths except those which originate in close proximity to the lung and invade that structure, producing early respiratory symptoms. These tumors seldom produce pain early in their course (see Group IV described below).

**4. Pressure Effects.**—We have spoken of those effects which result when the tumors originate from or encroach upon the bronchial tree, especially the large branches. Bronchial dilatation of varying degree may result. This may account for the paroxysmal coughing fits with “unloading” of considerable sputum which some patients complain of. It is the most reasonable explanation of the associated clubbing of the fingers seen in some patients. Sooner or later in most of the patients pressure effects are evident. In those tumors originating near the periphery of the lung pressure effects occur only late in the disease, while those originating from the mediastinum show pressure effects relatively early. Pressure symptoms, therefore, are variable, and when they do occur are in many ways comparable with those of mediastinal tumors. Changes in the voice due to involvement of the recurrent laryngeal nerve, dyspnea, prominence of the veins in the neck and upper chest, cyanosis of the face, edema of the arm, difficulty in swallowing, difference in the radial pulses, etc., have all been noted.

The **general symptoms** are those of malignant disease occurring elsewhere in the body. Progressive weakness, loss of weight, and secondary anemia develop in the course of the disease. There are cases in which the constitutional symptoms are more marked than the local symptoms and may even antedate local symptoms. This is particularly true of the deep-seated primary growths. That the so-called malignant cachexia does not invariably occur is illustrated by one of Adler's patients, who kept stout, florid, and apparently without any loss of strength until his death, which was caused by suffocation due to a profuse and sudden hemor-

rhage. However, progressive weakness and anemia are the rule. The blood count shows nothing of significance other than the progressive reduction of the hemoglobin and red cells. A leukocyte count in the neighborhood of 15,000 to 18,000 has been reported in a number of cases, and in a few instances where a high leukocyte count occurred there was in all probability an associated extensive necrosis or complicating infection.

In most instances *fever* of varying grade is present. It is usually intermittent, and if the neoplasm is not complicated by some associated condition the febrile disturbance is slight, but when associated with bronchitis, bronchiectasis, necrosis of growth, etc., it may be quite marked. In acute, rapidly spreading disease fever is more apt to be present than in chronic slow-growing neoplasms.

Symptoms referable to various organs distant from the lungs occur when metastases result. Adler, in his monograph, tabulates the frequency of metastasis to the various structures. Among 374 cases of carcinoma the lymphatic metastases predominate; of the other tissues, the liver was involved in 103 cases, kidneys in 58 cases, central nervous system in 57 cases, spleen in 18 cases, thyroid in 12 cases, pancreas in 6 cases. Among 90 cases of sarcoma the lymph-nodes were involved in 55 cases, the liver in 16 cases, pericardium and heart in 9 cases, bones in 8, central nervous system in 6 cases, pancreas in 5, spleen in 5. Metastases were absent in 33 cases of carcinoma and 15 cases of sarcoma.

In a given patient who presents local symptoms referable to the lung and local symptoms referable to some other organ—*e. g.*, the stomach—the question arises as to which is the primary growth. A careful study of the history and onset of symptoms will aid greatly in distinguishing the primary site. Occasionally, however, the rapidly growing secondary neoplasms in the lung may so overshadow the symptoms of the primary growth elsewhere that the lung tumor may seem primary. Thus Boris' patient presented all the symptoms of primary malignant disease of the lung, but at autopsy the microscopic examination revealed a chorionepithelioma secondary to a small, scarcely

perceptible focus in the broad ligament. Lesieur and Raue reported a similar case with massive involvement of the left lower lung, giving the clinical appearance of a primary growth. Autopsy revealed a small primary growth in the rectum which had given no evidence of its presence during life, and which was found to be microscopically the same as the lung growth (cylindric-cell carcinoma).

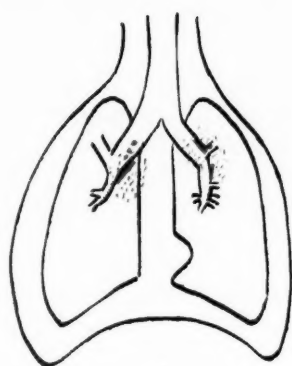
In order to facilitate a clearer understanding of the varying symptomatology of malignant growths of the lungs an arbitrary grouping of the patients into five groups has been made based upon the point of origin of the neoplasm. A knowledge of these groups will explain the varying sequence of symptoms in different patients, and will lessen the confusion which surrounds any effort to picture a composite case of pulmonary neoplastic disease. These groups I have charted as follows:

#### GROUP I

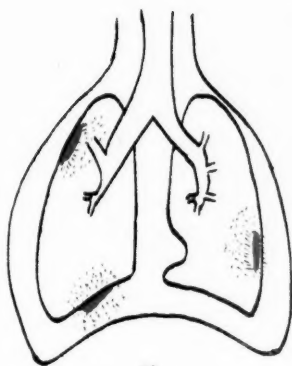
Tumor originating in or near the main bronchial tree. Cough and expectoration are early and the symptoms are not unlike those of ordinary subacute and chronic bronchitis. Blood spitting is frequent and may be profuse. A frank hemorrhage may be the first evidence of trouble. Dyspnea usually is early, although if bronchial obstruction is gradual and the other lung functionates properly, dyspnea may not occur till late in the disease. Pain is infrequent. The signs are frequently unilateral, diminished expansion, distant breathing. A slow-growing tumor gradually obstructing the bronchial lumen may give rise to bronchiectasis, paroxysmal cough, profuse expectoration, and clubbing of fingers. This type is frequently mistaken for pulmonary tuberculosis. The sputum, however, remains negative for tubercle bacilli. It may contain cell masses suggesting neoplasm. The x-ray and the bronchoscopic examinations determine largely the diagnosis.

#### GROUP II

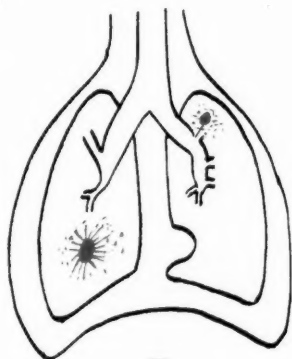
Tumor originating in or near the pleura. The symptoms and signs are those of pleurisy with effusion, or of chronic hyper-



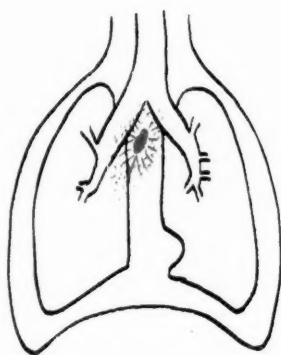
I



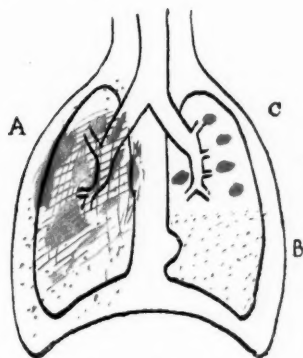
II



III



IV



V

Figs. 230-234.—Illustrating groups I to V (see text).

plastic pleuritis. Pain is early, may be severe and acute, or mild and recurrent. In some cases from the start, in others following the acute pain, there exists a constant, dull, neuralgic pain, especially when the tumor extension infiltrates the chest wall. The referred pains to the shoulder, down the arm, and into the abdomen are most frequently found in this type. Cough is an early symptom, but it is unproductive and may disappear. It is not productive until late in the disease, when the tumor has extended considerably beyond its point of origin. Blood spitting occurs only late in the disease. Dyspnea is a fairly early symptom and may be ascribed to the pain, encroachment upon free movement of the lung by the accumulation of fluid in the pleural cavity or fixation of lung to the chest wall, and interference with free movement of the diaphragm. The physical signs are usually those of thickened pleura or of effusion. Paracentesis thoracis reveals a small or large quantity of reddish-brown fluid which is sterile. The x-ray is a decided help in diagnosis.

### GROUP III

Tumor originating from pulmonary alveola forming deep-seated growth distant from main bronchial branches and from the pleura. Tumor symptoms and signs may be latent until growth has attained considerable size or metastasis has occurred. This type in some instances may be discovered only during the course of routine x-ray examination of chest. Progressive weakness and the constitutional symptoms of malignancy may antedate the local respiratory symptoms. Dyspnea, in part due to weakness, mediastinal metastasis, and the encroachment upon the respiratory surface is oftentimes the earliest local symptom. Cough, expectoration, blood spitting, and pain are late symptoms, and when these occur there are usually signs of marked involvement of the lungs—distinct limitation of movement, distant breath sounds, many or few râles. If the lesion involves the upper lobe the diagnosis of advanced tuberculosis is suggested, but the sputum is negative for tubercle bacilli. Lues may be suspected, but the Wassermann is negative. The x-ray reveals a definite, deep-seated involvement.



## GROUP IV

Tumors originating in the mediastinum, or from one of its structures, and extending to the lungs. This type gives rise to symptoms that are usually characterized as pressure effects, and the symptoms and signs of involvement of the lungs develop afterward. It is not our purpose at this time to discuss mediastinal tumors, except those which originate so near the hilus of the lung as to give rise to early symptoms and signs referable to the respiratory tract. This latter variety of Group IV then approaches Group I. The pressure effects which result from mediastinal tumors vary with the anatomic relation and rapidity of growth. Pain is usually present and a prominent early symptom, but it may be absent. When present it may be referred to the front or back of chest. It has been variously described as dull or aching; a feeling of discomfort, of pressure, and at times lancinating. It is not infrequently paroxysmal. There may be dysphagia. Dyspnea occurs when the trachea or primary bronchi are involved, and, once established, it becomes increasingly severe. Stridor is not infrequent. Cyanosis is in direct proportion to the respiratory difficulty. Cough, expectoration, and blood spitting follow. The cough frequently resembles the distressing, rasping cough of aortic aneurysm. Tracheal tugging may be found and dilatation of veins over chest, neck, and face is in evidence. The diagnosis is verified by the x-ray, although the history and Wassermann test must be studied to eliminate lues.

## GROUP V

The miscellaneous group comprises:

(A) One large or a number of small coalescing metastatic growths which involve the pleura, lung, bronchial tree, and mediastinum, singly or in combination, and present the clinical picture of Groups I, II, III, and IV in varying degrees. The clinical manifestations of the primary growth are at a distance—*e. g.*, the long bones, etc. (sarcoma), or the prostate, stomach, rectum, breast, etc. (carcinoma). Occasionally such a growth may be so rapid and prominent as to overshadow the primary growth and give rise to the belief that the pulmonary lesion is primary.

(B) Acute miliary malignant disease which runs a course not unlike that of acute miliary tuberculosis. There are small nodules, which look not unlike miliary tubercles, scattered over one or both lungs. It is usually secondary to a lesion elsewhere in the body, or perhaps in the lung itself. Elsberg reports such a case in which the primary growth was in the bronchus. Cough, dyspnea, cyanosis, rapidly progressing weakness, and signs like those of bronchopneumonia are present, the patient dying within a few weeks or months.

(C) A few scattered metastatic growths giving rise to no symptoms or signs and discoverable only at autopsy. The metastatic growths of this subtype are usually few, small, deep seated in pulmonary parenchyma, and away from the pleural and bronchial tree.

#### REFERENCES

- Adler: Primary Malignant Growths of the Lungs and Bronchi, 1912, 1st ed., Longmans, Green & Co., New York and London.  
Ash: Jour. Amer. Med. Assoc., 1915, lxiv, II.  
Aufrecht: Nothnagel's Encyclopedia, Diseases of Bronchi, Pleura, and Lungs, 1902, 713.  
Boris: Arbeit. Path. Anat. Inst., Tübingen, vi, Ht. 2, p. 539.  
Elsberg: Neoplastic Diseases, 1919, 1st ed., 786.  
Fowler and Godlee: Diseases of the Lungs, London, 1898.  
Garre: Verhand. des deutschen Geiellschoft fur cher., 1919, 1, 121.  
Guyot and Parcelier: Revue de Chirurgie, 1912.  
Kuttner: Congress de Chirurgie, 1908.  
Lesieur and Raue: Lyon Medicale, 1909, cxiii, 74.  
McCrae and Funk: Jour. Amer. Med. Assoc., 1919, lxxiii, 161.  
Passler: Virchow's Archiv., 1896, Band, cxiv, 5191.  
Rolleston and Trevor: Brit. Med. Jour., Feb. 19, 1903.  
Sailer and Torrey: Penna. Med. Jour., 1913, xvi, 539.  
Seydel: Münch. Med. Woch., 1910, lvii, 9.  
Stevens: Amer. Jour. Med. Sci., 1912, cxliv, 193.  
Stokes: Diseases of the Chest, London, 1837.  
Wolf: Fort. d. Med., 1895, xiii, 725.  
Woolley: Amer. Jour. Med. Sci., 1903, cxxv, 33.

## CLINIC OF DR. MARTIN E. REHFUSS

JEFFERSON HOSPITAL

### ANALYSIS OF DISEASES OF THE GALL-BLADDER AND DUCTS

Anatomic and Physiologic Relations; Pathologic Considerations; Cholelithiasis and its Clinical Manifestations; Position and Manifestation of Stone; Diagnosis of Gall-bladder Disease; Differential Diagnosis; Chemical Examination of Bile; Full Description of Various Tests for Bile and Bile Acids; Indirect Analysis of Bile; Correlative Diagnosis; Importance of the Study of All the Methods Herein Described in Every Case of Chronic Gastro-intestinal Disease in which there is a Suspicion of Gall-bladder Trouble.

#### ANATOMIC AND PHYSIOLOGIC CONSIDERATIONS

A BRIEF consideration of the anatomy and physiology of the gall-bladder and biliary passages is in order before we shall attempt to discuss in detail the method of analysis to be pursued in the study of these organs.

The biliary passages, including the gall-bladder, represent the system for the storage and disposal of the bile after it has been formed in the liver. By means of this apparatus, as Meltzer has pointed out, *we have the transformation of a continuous into a discontinuous flow*. In other words, the liver which forms bile more or less continuously must have some method by which this bile can be stored up until it is needed, and it is now a recognized fact that when the intestinal tract is in need of bile the quantity which is essential for perfect digestion is more than can be secreted by the liver during that interval. The first important principle to be remembered is the fact that anatomically and embryologically the liver, gall-bladder, bile-passages, duodenum,

and stomach are intimately correlated and are all parts of the primary entoderm forming the alimentary canal. The original liver develops as a diverticulum from the duodenum, and the gall-bladder and bile-passages develop as diverticulum from it. Furthermore, we know that these organs receive the same nervous enervation. Fibers of the splanchnic nerves through the celiac axis supply the stomach, duodenum, pancreas, gall-bladder, bile-ducts, and liver, while the same is true of the vagus. Experimental stimulation of the splanchnics inhibits the flow of bile, while section increases the flow. MacCarty struck the keynote when he said that these organs must not be functionally separated, but could be considered even pathologically as a gastroduodenohepaticopancreatic physiologic system. This fact accounts for the similarity of many gastric, pancreatic, and biliary syndromes, and explains the difficulty in dissociating these cases except by careful analysis. Similar embryology, similar enervation, and finally, similar blood-supply offer an excuse for the diagnostic difficulties which arise.

Briefly, the biliary system consists of an hepatic duct which is made up of two branches, a right and left, emerging from the respective lobes of the liver. These collecting ducts unite to form the *hepatic duct which runs along the free edge of the gastro-hepatic omentum* in front of the portal vein and to the right of the hepatic artery. It is about 1 inch or more in length and from 4 to 6 mm. in width. It then joins the cystic duct from the gall-bladder to form the *common hepatic duct*.

The common bile-duct then descends, crossing the posterior surface of the pancreas, which it either indents or in which it forms a complete canal, and finally ends in the junction of the first and second portion of the duodenum or just below this point. Near its termination it meets the pancreatic duct and with this duct pierces the duodenal wall obliquely to empty into the duodenum by means of a well-marked ampulla known as the ampulla of Vater. Its length is about  $2\frac{3}{4}$  inches and its diameter 6 mm. The *gall-bladder* represents a lateral diverticulum or reservoir which lies in a special fossa in the inferior surface of the liver. It is from 3 to 4 inches in length and contains about 50

c.c. ( $1\frac{1}{2}$  ounces—Piersol). It is a pear-shaped organ narrowing to a terminal portion or neck, which constitutes the *cystic duct*. It is attached to the liver directly without the interposition of peritoneum, which simply covers the unattached surface, and in health rests on the transverse colon and the first part of the duodenum.

Several important anatomic considerations must be borne in mind. In the first place, the presence of valves which mark the cystic duct and also the presence of the sphincter of Oddi, which marks the termination of the common duct, are supposed to be usual points for the arrest of stone. Again, the more or less parallel arrangement of the cystic and hepatic ducts explains why a stone in the former can compress the latter, inducing obstructive jaundice. Also the intimate association of the gall-bladder, duodenum, and pylorus emphasizes the many disturbances which occur to the latter from inflammatory bands, etc. Furthermore, it must be remembered that the association of the duct of Wirsung of the pancreas and the termination of the common bile-duct can explain either a simultaneous ascending infection of both ducts from the intestine, or an ascending infection of the pancreas from an associated biliary infection.

Histologically the gall-bladder is a fibromuscular organ containing smooth muscle-fibers and also a mucous membrane covered with simple columnar epithelia. The structure of the ducts is similar, but contains little muscle structure. Physiologically, according to Meltzer, there are two sets of muscle-fibers, one in the gall-bladder and one at the end of the common duct, the so-called Oddi sphincter. When the latter is contracted, the musculature of the gall-bladder is relaxed, and owing to the increase in pressure of the bile in the ducts the bile is diverted into the gall-bladder to be used in response to certain stimuli. These stimuli, which are supposed to be the peptones and fat, produce, by crossed enervation, contraction of the gall-bladder and relaxation of Oddi's sphincter, with the emission of bile into the intestine. There can be no doubt that the bile is under pressure in the common duct, and this pressure increases with the contraction of the sphincter.

The secretin (Bayliss and Starling) formed in the duodenum excites at the same time the pancreatic and biliary secretion. *It must be remembered that in man, unlike in certain animals, there is an intermittent flow of bile into the bowel, and this conversion of a continuous flow from the liver into a discontinuous flow is furnished by the gall-bladder.* The importance of this mechanism is readily seen by the frequency with which diverticula form after cholecystectomy.

**Bile** represents an excretion of the liver. It serves as adjuvant to digestion and also a means for the elimination of toxic substances on the part of the liver. Chemically, it is 90 to 95 per cent. water, 5 to 10 per cent. bile salts, 1 to 2 per cent. bile pigments, cholesterol, mucus, etc. The *bile salts* are the sodium taurocholate and glycocholate nitrogenous products in which cholic acid is combined with amino-acids, glycoll, or taurin. These substances do not exist in the blood, but are formed by the liver cell; they are produced by the breaking down of protein and are very toxic (Landouzy). The *pigments* are derived from hemoglobin. Practically all the pigment formed by the liver is eliminated in the bile and is likewise toxic; in fact, the whole bile is toxic, nine times as toxic as urine (Bouchard). A very small amount of the bilirubin formed passes into the blood and is eliminated by the urine after having been transformed into urobilinogen. *Cholesterol* is also part of the bile formed and is secreted in the liver. It is a lipoid, partly exogenous in origin from the food ingested and partly endogenous in origin from the formation of cholesterol in the liver. It is held in solution in the bile owing to the presence of bile salts. The importance of this constituent will be discussed later.

Bile is of importance in *the emulsification and absorption of fats*. In fact, fat digestion is largely due to biliary and pancreatic activity. In biliary retention the stools become white, greasy, and fat absorption is markedly reduced. Furthermore, the bile serves as a means of elimination not only of many toxic substances but also of bacteria (Richt).

An interesting question which arises is, What becomes of the bile, part of which must be absorbed? Part of the bile salts and

pigments are decomposed by bacterial action and ferments and are eliminated in the feces (Gilbert and Hershercit, Landouzy) under the form of cholic acid, taurin, and glycocoll for the former, and stercobilin (a substance identical to urobilinogen) for the pigments. Part, however, is reabsorbed, taken to the liver, and re-eliminated, constituting what the French call the "entero-hepatic circulation." In fact, we are told that the bile absorbed constitutes the best bile stimulant and offers the explanation for the logical use of bile internally.

#### PATHOLOGIC CONSIDERATIONS

**Gall-bladder.**—The gall-bladder is subjected to the same pathologic processes as other organs of the gastro-intestinal tract, with the exception that owing to its peculiar function, and the tendency for the stagnation of altered bile, we have also calculous formation as an entity. In other words, we have acute and chronic inflammations; more rarely, the systemic conditions, like tuberculosis, syphilis, and actinomycosis; new growths, of which the benign sorts, such as cysts, adenomata, and papillomata, are rare, and the malignant tumors, such as sarcoma and carcinoma, of which the latter is more frequent. Traumatic rupture of the gall-bladder is rare, but perforation often occurs, especially in empyema.

**Acute cholecystitis** is nearly always due to bacterial infection of the gall-bladder. Less frequently toxins reaching the organ are given as the cause. As shown in Fig. 235, infection occurs in one of three ways: (1) By way of the blood-stream, or hematogenous, in which through septicemias and pyemias—not infrequently in pneumonia and influenza—the organism is carried directly to the gall-bladder. This mechanism includes the so-called elective localization of organisms (streptococci—Rose-now) in the gall-bladder wall; (2) by an ascending infection from the intestinal tract (duodenitis); (3) by the elimination of the organisms in the bile and their storing up in the gall-bladder—the colon influenzal and typhoid groups are supposed to act in this way. This seems to be the most logical explanation of the presence of the typhoid organism, namely, through the por-

tal system, and finally into the bile. The *bacteriology of acute cholecystitis* represents not merely the colon and typhoid group, but the paratyphoid (Cecil), the streptococcus, staphylococcus, and pneumococcus. In our experience the colon and the staphylococcus are the organisms encountered most frequently,

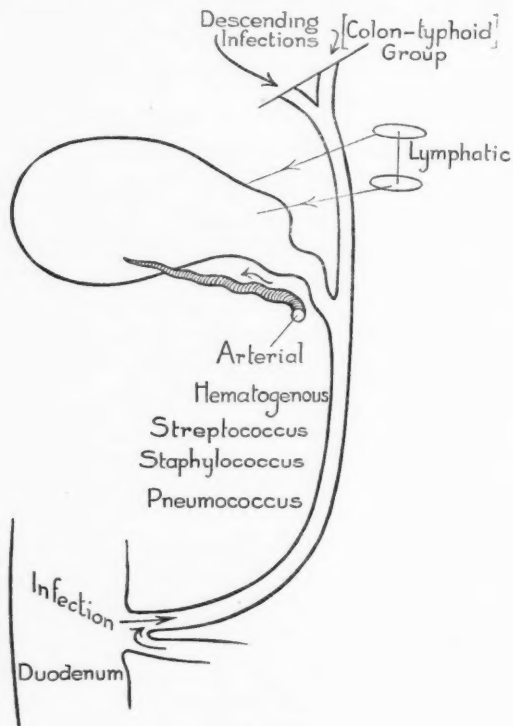


Fig. 235.

although Adami claims that the colon organism can be eliminated by the bile in health.

The forms of acute cholecystitis depend on the extent and severity of the lesion, and are classed as acute catarrhal, membranous, suppurative, phlegmonous, and gangrenous. Symptoms are:



(1) Generalized abdominal pain due to the association of the branches of the celiac axis and the vagus.

(2) Pain over the gall-bladder area at the intersection of the right costal cartilage (ninth) and the border of the right rectus.

(3) Not infrequently referred pain to the right shoulder owing to the association of the phrenic and supra-acromial nerves through the fourth cervical plexus.

(4) Upper right rectus rigidity, owing to the association of the splanchnics and the intercostals (White).

(5) Nausea and vomiting due to the splanchnics and vagi, later through toxemia and peritonitis.

(6) Localized tenderness over the gall-bladder area.

(7) If there is peritonitis, the general signs of that condition.

(8) Leukocytosis if there is inflammation with pus.

*Differential Diagnosis.*—This must be made from all acute abdominal conditions, but particularly: (a) appendicitis, in which the localization of the pain and the maximum tenderness is different; (b) acute hepatitis, in which the pain is more diffuse and the liver enlarged, this includes abscess of the liver; (c) pyelonephrosis (urinary findings), in which the urinary phenomena and the lumbar pain serve to differentiate the condition; (d) subphrenic abscess, in which the symptoms are preceded by the causative factors. A differential diagnosis of the form of cholecystitis is likely by the following symptoms: the acute form with calculus is nearly always preceded by biliary colic and the antecedents of stone; in the catarrhal form there is some tenderness, little fever.

Various forms of acute cholecystitis occur: the *acute catarrhal type* is that which stops short of pus production. It is accompanied by local pain and tenderness, spasmodic pain less severe than colic due to cramp of the musculature, cutaneous hyperesthesia over the ninth and tenth dorsal segments, rigidity of the upper right rectus, sometimes distention of the gall-bladder and vomiting either through irritation or actual peritonitis. *Pericholecystitis* may occasion the signs of local peritonitis, and remotely, intestinal obstruction, as well as produce wide-spread adhesion formation to the duodenum, pylorus, and adnexia.

A *membranous* form and an *acute hemorrhagic* form have been described; if the condition goes on we may get *suppurative cholecystitis* characterized by pain, tenderness, rigidity, and all the common signs of pus—chills, fever, leukocytosis, etc. Jaundice is usually absent unless there is associated angiocholitis. Leukocytosis varies from 15,000 to 30,000 (Rolleston), but may not occur in a pure typhoid infection. The chief danger of this form is *ulceration* and *perforation* with general peritonitis. Perforation may form localized abscess, distinguishable with difficulty from subdiaphragmatic abscess due to disease of the upper digestive tract. It must also be differentiated from perforating duodenal ulcer and an abnormally placed appendix. *Gangrenous cholecystitis* is rare and resembles acute perforative peritonitis in the upper abdomen.

**Chronic cholecystitis** is nearly always due to repeated acute attacks, although in certain instances the disease occurs from the start as a chronic, persistent infection of low virulence. Most frequently it is associated with calculous formation and the two processes go hand in hand. Any number of forms are found; the "strawberry" gall-bladder described by MacCarty as chronic catarrhal cholecystitis, owing to its peculiar appearance (mucous membrane resembling strawberry seeds), to forms in which the predominating features are atrophy and adhesion formation (pericholecystitis), producing considerable deformity in the adjoining organs. The *diagnosis* may be based on colic attacks (stone) with persistent local tenderness, or again, on the evidence of local physical signs during an acute exacerbation. In many of these cases the tenderness is more or less constantly present. These are the types associated with chronic appendical disease, and they are the class *in which the gall-bladder infection is occasionally due to primary infection elsewhere*. At least this possibility should be borne in mind in dealing with these cases.

**Systemic Diseases Involving the Gall-bladder.**—Tuberculosis has been described as being responsible for the fistula accompanying cholecystotomy; its presence in the gall-bladder is rare, however. The same may be said of actinomycosis and syphilis.

**New Growths.**—Cysts, adenomata, and papillomata have

been described, but belong to the category of rather infrequent conditions. Sarcoma has been described, but the most frequent form of malignant disease of the gall-bladder is *carcinoma*. Carcinoma of the gall-bladder is not rare. It occurs most frequently in the fundus which is exposed most to calculous action, but may occur in any portion of the gall-bladder. When it occurs at the neck of the bladder or the cystic duct it can cause enlargement and distention of the organ. It affects the liver in 50 per cent. of cases, but metastases may occur throughout the abdomen through the abdominal lymphatics, or again in the chest, involving the lungs, or, even more rarely, enlarging the lymphatic glands above the clavicle.

An important consideration is the association of gall-stones with carcinoma of the gall-bladder. In from 70 to 90 per cent. of the latter cases gall-stones are found, and the belief is held by many that calculi are responsible for the condition. The diagnosis is made by the pressure of a hard, nodular tumor in the gall-bladder area, progressively increasing in a woman, in most instances after middle life. With this is approaching cachexia, anorexia, and dull pain in the gall-bladder region. The tumor must be differentiated from other upper abdominal tumors, such as movable kidney, pancreatic cysts, benign enlargements of the gall-bladder, and tumors of the stomach, all of which are capable of confusing the condition. The frequent association of cancer of the liver will often mask the condition. The cancerous nature of the tumor is frequently shown by the progressive emaciation and cachexia, the increasing anemia, the continued pain, and the hardness of the tumor. The association of a previous gall-stone history will sometimes aid in differentiating carcinoma of the gall-bladder from carcinoma of the liver. Occasionally carcinoma of the gall-bladder may remain latent and the symptoms be entirely due to secondary manifestations.

#### CHOLELITHIASIS

In the study of gall-stones we must carefully consider: (1) The etiology of the condition, (2) the clinical manifestations giving varying syndromes, depending on the localization and

extent of stone formation, (3) the attitude to be adopted toward the presence of calculus.

**Etiology.**—Today we believe that the mechanism of stone formation can be grouped under three heads: (1) Stasis of bile, (2) infection of bile, (3) alteration in bile. We realize that stone formation, while it takes place in the great majority of cases in the gall-bladder, may nevertheless occur in the hepatic duct or even in the smaller biliary radicles of the liver. I have obtained cholesterol concretions from the substance of the liver itself. *Infection* is considered by many to play the predominating rôle, and the typhoid bacillus was held for a long time responsible for this change. Chauffard showed, however, that the incidence of typhoid fever in individuals who had stones was not very much greater than in those without manifestations of calculous formation. Furthermore, we know that there is another mechanism besides infections which plays a rôle in the formation of stone after typhoid fever, namely, the hypercholesterolemia. Infection acts, as we have already pointed out, in acute cholecystitis, either through the blood-stream by ascending infection, or through the mechanism of elimination on the part of the liver. Stone formation, however, is nearly always accompanied by evidences of infection, but infection is by no means always accompanied by evidences of stone formation. The organisms concerned in this change have been mentioned under the heading Acute Cholecystitis. *Stasis* acts principally by allowing the bile to reach undue concentration and also by virtue of the fact that in many instances bile is excreted in which the cholesterol present is barely held in solution by the bile salts present. The mechanism of stasis is seen in a new light when we consider the mechanism of "contrary innervation," by which during the rest periods the bile is stored up in the gall-bladder by the contraction of Oddi's sphincter and the relaxation of the walls of the gall-bladder. Meltzer pointed out that in dietary irregularities the bile may be stored up for a long time in the gall-bladder and lead to precipitation. At least in those cases where the bile is manifestly altered irregular eating could be considered as a possible source of stone formation. *Alteration in bile* is one

of the most important causes of stone formation. In other words, instead of a fluid, limpid bile, the bile excreted is altered in consistency and also in its constituents, particularly *cholesterol* and *bile salts*. Anyone who has done much duodenal work will be convinced of the alterations which occur in bile, and I have on many occasions observed material which was obviously different from the normal material. The most important element, however, as Chauffard pointed out, is cholesterol, and the mechanism by which this substance acts is, briefly, as follows: Cholesterol occurs in the blood-serum in health in a concentration of 160 to 180 mg. per 100 c.c. of serum. Its origin is both exogenous from cholesterol rich foods, and endogenous from the activity of certain cholesterogenic organs, such as the suprarenals and the corpora lutea. Both of these mechanisms are responsible for it, although on various occasions one or the other is in preponderance. Under certain conditions the amount of circulating cholesterol can markedly increase; this is seen in the convalescent stage of typhoid fever, but is not alone confined to this disease, being also seen with the exanthemata; in the later months of pregnancy and after the first few days following delivery there is pronounced hypercholesterolemia; to a lesser degree it is seen in chronic degenerative conditions, such as nephritis and arteriosclerosis, also in syphilis and diabetes.

The next step in the chain was to prove the association of an increase in blood cholesterol with the concentration of the cholesterol in the bile, and this was done by McNee and Pierce. Chauffard points out, however, that in cholelithiasis the real offender is the "liver cell." Owing to the constant absorption of toxins via the portal area from constipation, etc., there results an alteration in the bile excreted, in which there is present not merely an undue concentration of cholesterol but also an insufficient amount of bile salts to hold this substance in solution. The result is, particularly if there is also stasis and infection, calculous formation. The question which is naturally of interest is the incidence of hypercholesterolemia in demonstrable stone. In our experience 80 per cent. or more of operable stones show an increase of cholesterol in the serum. This does not prove,

however, that at the time of stone formation all cholesterol stones are accompanied by this finding. In many instances the active period of stone formation is over when the patient undergoes operation; furthermore, not all stones are cholesterol stones. In certain instances stasis and infection seem to play the predominating rôle, but neither stasis nor infection is possible without alteration in bile and its constituents. The so-called cholesterol diathesis occurs in certain families which are particularly predisposed, and may be mentioned as a possibility similar to the uric acid or gouty diathesis.

**Clinical Manifestations of Cholelithiasis.**—*Number, Size, and Position of Stones.*—They may vary from one to many thousand stones. The largest number on record is that reported by Otto (Roleston), 7802. The largest number that I personally have encountered was 734 stones in a gall-bladder which was prolapsed and as large as a child's head. Most stones are faceted or rounded off, although occasionally a mass of concretions, usually belonging to the pigment variety, may show no sign of faceting, but be sharp and prickly. In Kelly's analysis (System of Medicine, Osler and McCrae, 1908, V, 831) the position of the calculi was: 55.5 per cent. in the gall-bladder alone, 12.5 per cent. in the gall-bladder and cystic duct, 10.5 per cent. in the gall-bladder and common duct, 6.6 per cent. in the common duct alone, and 6 per cent. in the cystic duct alone. Not infrequently a stone may be found buried in a diverticulum of the gall-bladder in which presumably the ulceration induced by the stone has brought about weakness of the gall-bladder wall.

**Signs and Symptoms of Stone.**—Gall-stones give varying symptoms, depending on the position of the calculi. In fact, the simplest way in which to approach the problem is to consider the stone from the standpoint of its position. In this way we have four separate syndromes differing in their clinical pictures (Fig. 236):

1. Stone in the gall-bladder.
2. Stone in the cystic duct.
3. Stone in the common duct.
4. Stone in the ampulla of Vater.

These syndromes may exist alone or, as has been pointed out above, several combinations may exist together, as, for instance, stones in the gall-bladder with a stone in the cystic duct or common duct. The important point to bear in mind, how-

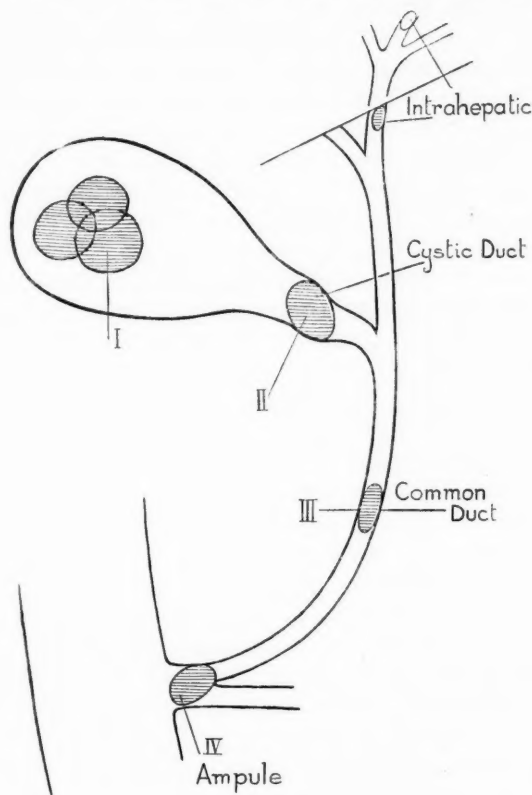


Fig. 236.

ever, is the mechanism by which these different forms act. Stone in the gall-bladder acts *reflexly* and not mechanically; therefore there are no obstructive phenomena, and unless there is pronounced irritation and infection of the gall-bladder wall there is no colic. In this group all the phenomena are digestive,

reflected over the pathways of the vagi and the sympathetics. It is this group which long before it strikes by migration of the stone—producing colic—induces all sorts of digestive disturbances, frequently masquerading under the clinical picture of ulcer or chronic appendicitis, making an accurate diagnosis very difficult. Aërophagia, vagotonia, and pylorospasm are the commonest manifestations, and the belching, eructations, fulness, and dull pain are characterized principally by their atypical time and unusual combinations in an individual who usually is, as Deaver would say, “fair, fat, and forty or over.” These dyspeptic symptoms are the so-called “inaugural” symptoms of Moynihan. I have examined many of these cases, and there is no fixed rule so far as their gastro-intestinal manifestations are concerned. While Ohly found hyperacidity in 18 out of 77 cases, in 46 HCl was either lacking or deficient, and in 13 normal secretory findings were present. In my experience more than one-half show hypersecretory findings, while the low findings frequently associated with evidence of gastric infection are to be found in old lesions, principally those associated with a duodenitis or a gastric catarrh.

Stone in the *cystic duct* produces biliary colic, but of the non-obstructive variety so far as the flow of bile is concerned. Occasionally, owing to the more or less parallel arrangement of the cystic and hepatic ducts, stone in the former may compress the latter. Cystic duct block reacts more on the gall-bladder than it does on the excretory mechanism. Stone in the *common duct* produces the typical colic associated with obstructive jaundice. In other words, the typical form of biliary colic is seen in the latter condition. Stone in the ampulla of Vater is not infrequently associated with pancreatic disturbances.

#### STONE—POSITION AND MANIFESTATIONS

	Gall-bladder.	Jaundice.	Urine.	Feces.	Remarks.
Gall-bladder....	Some cases enlarged.	0	0	0	Reflex.
Cystic duct....	Enlargement.	0	0	0	Simple colic.
Common duct ..	+ and -.	+	+	Acholia.	Colic jaundice.
Ampulla .....	+ and -.	+	+	Acholia.	Associated pancreatic.



Biliary colic is probably the most typical manifestation of stone. It is commonly attributed to spasm of the ducts or even spasm of the gall-bladder wall. Some clinicians attribute the pain not alone to the mechanical action of the stone and spasm, but to the associated infection or cholecystitis which occurs. Although this might induce painful contraction of the gall-bladder or ducts, there is no reason to doubt the belief that typical colic is induced, and it is now believed that the pain is due to spasm plus inflammation. The pain of biliary colic is extremely severe, and the picture as it most commonly occurs is somewhat as follows:

The patient is seized usually at night between the hours of 11 to 2—but by no means confined to these hours—by severe paroxysmal and lancinating pain in the epigastrium and through the upper right quadrant of the abdomen. It radiates in all directions, to the right side or the left, through to the back, so that the patient feels as if the back were broken, throughout the abdomen frequently from side to side, often to the right shoulder, and not infrequently radiating through the chest and down one or both arms, resembling an angina. The pain is excruciating and may occur at any time. One of my patients was seized in an elevator, another while calmly reading the paper and resting, many while engaged in some physical labor, but by far the vast majority are seized at night and four, five, and six hours after meals, when the gall-bladder makes its most intense expulsive effort. On a number of occasions I have been up all night with these cases watching the evolution of the condition. Nearly always the patient is tremendously excited, and his abdomen is distended with gas due to simple aërophagia. In fact, this aërophagia of the acute type is one of the typical manifestations of the attack. The patient, unlike the individual with acute inflammatory disease, such as peritonitis, may pace the floor, double up his fists and press them into the abdomen, double himself over the back of a chair, or roll from side to side in great pain. Pressure on the abdomen in most cases seems desirable, and the patient is most frequently seen with his fists tightly compressed against the abdomen; not infrequently he will com-

plain of intense backache. Usually there is no hesitation in the physician's mind as to whether morphin ought to be given; it has to be given because the patient cannot stand the pain any longer. Instances have been recorded in which patients died from the shock of the condition. I have seen them with sweat rolling off their forehead in utter mental and physical agony. Very frequently the first injection is not sufficient and a second or third may be necessary. Very early is seen the indication to relieve the bowels and even to perform gastric lavage, although an attack of vomiting may initiate the latter. The great distention with air in many instances is synchronous with the marked discomfort, and relief comes with the removal of these aërophagic crises. Gradually the pain lessens and leaves in its track tenderness, usually over the gall-bladder area. Often in these cases the patient will remark, early in the morning, that the character of the urine has changed and become darker or even a mahogany color due to bile pigments. I have in a number of instances noted that while the material removed from the bowel during the attack was well colored with bile material or fecal material, which appeared after the attack, or even before pain was completely relieved, was already acholic. With this, in the typical case as the dawn approaches, the physician is confronted with a patient who will show undeniable signs of subicterus in the sclera or even general icterus.

Such is the evolution of biliary colic of which every modification can occur. It is claimed that more than half the cases show slight fever, owing, presumably, to concomitant cholecystitis or cholangitis, but it is rarely pronounced, and fever, when well established, is rather against than for stone attacks. The pulse is usually normal or but slightly increased, and there is present occasionally a slight leukocytosis from concomitant infection, although leukocytosis of any degree is rare. Accompanying the attack is vomiting, usually of the food eaten, and then bile, indicating that during the early stage of colic there is no obstruction to bile output. *Jaundice* is of frequent occurrence, although there are many cases in which jaundice will not be detected. Stone in the cystic duct will not be accompanied

by jaundice. Again, stone is not necessary to this syndrome, inasmuch as inflammation alone, preceding stone migration, may give rise to jaundice. Jaundice may appear within several hours or only after a day or two, and bile pigment is frequently found in the urine before there is manifest jaundice.

**Differential Diagnosis.**—This is to be made from (1) renal colic, (2) cholecystitis, (3) perforating gastric or duodenal ulcer, (4) pancreatitis, (5) intestinal obstruction, (6) angina pectoris, (7) appendicitis, (8) the various forms of peritonitis.

*Renal Colic.*—The radiation of the pain to the groin, frequently micturition, blood, pus, or albumin in the urine, tender kidney, and the x-ray findings distinguish renal from biliary colic.

*Cholecystitis* may occasion similar pain, but the pain is rarely so paroxysmal and severe, and there are usually found symptoms of local infection in this condition.

*Gastric and Duodenal Ulcer.*—This diagnosis is most difficult when it suggests perforation. The pain of both gastric and duodenal ulcer is more regular, periodic, and less painful than that of biliary colic, and of different regional distribution. With perforation, however, the early signs of peritonitis, the previous history suggesting ulceration, the early pulse rise, temperature, and local findings of peritonitis, all suggest ulcer.

*Pancreatitis.*—In the acute disease pain is as intense or more so, but the shock and collapse is even greater than in biliary colic; in fact, in acute pancreatitis there is early collapse. The pain is more directly in the epigastrium and there is diffuse deep-seated tenderness and resistance in that area.

*Intestinal Obstruction.*—While due to many things, this can occasionally resemble biliary colic, and the appearance is kept up by the low temperature and slight pulse changes. However, the evolution of the condition is entirely different, and the failure to move the bowels and the evidence of localized distention are not seen in biliary colic.

*Angina Pectoris.*—I have seen biliary colic closely resemble in its distribution the pain of angina pectoris, and I have seen both conditions in the same individual. The sequence of the two, however, is quite different, inasmuch as biliary colic almost

always, even in the absence of jaundice, leaves a tender gall-bladder as a sequel, while in the cardiac condition a study of that organ will demonstrate changes found in that disease.

*Appendicitis* gives the pain, tenderness, and rigidity in the lower right quadrant of the abdomen. It is nearly always preceded by epigastric pain, which later settles in the lower right side.

*Peritonitis* must be borne in mind and any causal factors investigated.

Lead colic, hepatic crises in tabes, twisted ovarian pedicle, gastric crises, all have been mentioned as important points to be remembered in differential diagnosis, but most of these conditions will be apparent upon careful examination.

#### DIAGNOSIS OF GALL-BLADDER DISEASE

In the diagnosis of gall-bladder disease we must take into consideration the etiology and history of the patient as well as the physical examination, which is capable of yielding considerable information. If these studies emphasize the necessity for more careful exploration of the gall-bladder, we have two means at our service: (1) the examination of the bile (*a*) by means of duodenal intubation in which the bile can be obtained, submitted to microscopic and chemical examination, and also by various means, to culture, and (*b*) the examination of the bowel movement for the evidence of bile output and also fat digestion; (2) *x*-ray studies to demonstrate calculus or enlarged gall-bladder.

In the consideration of any case of gall-bladder disease we ask ourselves first—after the evidence points to this organ—for the possible etiologic factors which might be responsible for disease. Septicemia, pyemia, and pneumonia can all, through hematogenous channels, induce acute disease of the gall-bladder; typhoid fever, both through the bacteriemia and the hypercholesterolemia following the febrile stage, can precipitate attacks of stone; pregnancy can act likewise through hypercholesterolemia; constipation and intestinal infections can act principally through the portal vein, inducing changes in the liver cell with a resultant change in the character of the bile eliminated. The

frequent association of appendicitis with cholecystitis is being pointed out more and more, and there are clinicians who claim that the appendicitis is responsible for the cholecystitis, and vice versa. I have been struck by the association of gall-stones with evidences of a profuse colon growth, both in the bile eliminated and frequently in the urine.

*It must be evident that the bile is formed in the liver, and the character of the bile will be largely dependent on the material brought through the portal system and the condition of the liver cell.*

**Physical examination** reveals a number of points regarding gall-bladder disease. It may reveal: (1) tenderness, (2) hyperesthesia, (3) rectus rigidity, (4) a palpable tumor.

*Tenderness* is generally most pronounced at the intersection of the ninth costal cartilage and edge of the right rectus. Not infrequently the contraction of the rectus will prevent successful palpation of the gall-bladder area. I have frequently resorted to a means of palpation in which, when the rectus is more or less contracted or in muscular individuals, the fingers are hooked under the rectus, taking advantage of the comparatively lessened resistance of the lateral muscles of the abdomen. This procedure will often demonstrate well-defined tenderness when the direct palpation fails to show any such findings. Murphy's method of palpation is simple and often efficacious: the patient, after having removed all clothing, is placed in a sitting position. The examiner stands behind the patient and places his right hand, with the palm flat on the abdomen, immediately below the ribs on the right side, while the patient takes deep breaths. After each expiration the examiner's hand approaches closer to the gall-bladder area until suddenly the patient arrests his respiration with an exclamation of pain. According to Schmidt, pain in gall-bladder disease is most frequently referred to the right nipple, back to the shoulder-blade, and often to the lumbar region. As Bevan points out, this is more likely to occur in associated hepatitis (peri-). In my experience the colic nearly always begins in the epigastrium and then may radiate anywhere. The right shoulder radiation is by no means constant. In many instances in which there is evidence of chronic chole-

cystitis there is apt to be general soreness over the lower ribs, so much so that on several occasions the condition was taken for a pleurisy. The residual soreness in the gall-bladder after an attack of colic is a point always to be sought after. The tenderness in neoplasm is more localized, there is more persistent, although not such severe, pain. In all cases where there is tenderness the condition of reflex rigidity of the recti muscles must be tested. In gall-bladder disease the upper right rectus will frequently be contracted, while the lower portion of the same muscle will be more or less flaccid; furthermore, there is usually a very pronounced difference between the two sides. In most inflammatory conditions of the gall-bladder deep breathing is painful, and marked bodily movements, such as bending, induce pain. Heads' zones of hyperesthesia for gall-bladder include the area between the tenth and twelfth thoracic vertebræ; again we are told that the area supplied by the eighth dorsal is the one most often responsible for the hyperesthesia.

1. **Examination of the Bile.**—In the chemical analysis of gall-bladder disease we attempt to determine the quantity and quality of bile output either directly, by duodenal intubation, or indirectly, by means of fecal analysis.

(a) *Direct Analysis of Bile.*—By means of duodenal intubation it is possible to obtain directly and without much effort pure samples of bile, as the secretion is expelled through the ampulla into the duodenum. There are several methods by which this is performed, and the material obtained is almost always a mixture of bile, duodenal and pancreatic secretions.

Over 80 cm. of the tubing is passed after the ingestion of bouillon, and the patient is turned on his right side with the hips slightly elevated and well rotated to the right. Sometimes active peristalsis can be more readily produced if the patient is active, namely, by getting up and walking around the room. I have used pretty nearly every method I have heard of for duodenal intubation, but one method will succeed in a certain case, while another will succeed better with another variety. I have used the Jutte tube, but the Jutte tube will only succeed in a certain group of cases as brilliantly as has been described. Regardless

of what form of tube is employed, the requisite amount of tubing is passed, and then at intervals aspiration is performed until the characteristic syrupy bile is obtained. At this point I usually allow the tube to drain. In this way large amounts of bile can be collected.

*None of the Aspirated Material Should Be Used for Culture.*—On the contrary, the long distal end of the tube should be allowed to hang over the edge of the bed while the bile drains off by siphonage. In this way the pure bile emerges untouched by external objects. After this drainage has occurred for some time

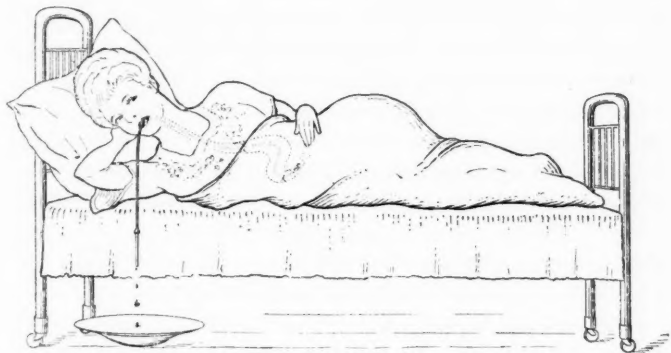


Fig. 237.

the technic can be altered by allowing a few drops of bile to drop into each of the culture-tubes. Blood-serum, agar, bouillon can be employed, and it is my custom to obtain at least three cultures separately. The value of solid media is the fact that the various colonies are well seen, while the liquid media, such as bouillon, require plating out. The criterion which we employ while not ideal, nevertheless enables us to form some conclusion regarding bacterial flora in the bile. If the bile is infected, *abundant cultures, practically pure in type, are seen in the various samples.* Accidental contamination usually yields discrete colonies. If, therefore, we obtain similarity in results with an abundance



of colonies, our inference is that the bile is infected. I have seen the freshly removed bile swarm with bacteria, a condition accounted for only by infection. These organisms are then submitted to separate study and identification. *The commonest organisms found in the bile are, first, the colon bacillus, then the staphylococci, less frequently the streptococcus, occasionally the pneumococcus and the Micrococcus catarrhalis.* Not infrequently organisms of the variety of the *Bacillus pyocyaneus* are found. I have cultured several hundred cases in this way, and our findings indicated that the organisms most frequently encountered are those mentioned above. Adami states that the colon bacillus eliminated in the bile can occur in health. That its elimination *en masse* is compatible with health I do not believe, and when it is present, which is very often, I attempt to define its pathologic significance by the correlative data of the case. In many of these cases I have used the colon bacillus for an autogenous vaccine with good results. In a certain proportion of cases I have found with a colon growth in the bile a colon bacilluria, in every case of which I used the colon vaccine. Most frequently in definite gall-bladder infection characterized by obvious physical signs there is associated a pus organism. The growth is then cultured and an autogenous vaccine prepared and administered according to the recognized rules governing the administration of this method of treatment.

*Centrifugalization and Microscopic Examination.*—While some observers have claimed that it is even possible to find cholesterol crystals in cholelithiasis, I have never seen typical crystals which could be diagnosed as those of cholesterol. I have, however, seen in angiocholitis well-marked pus in the bile, and in not a few cases I have found a definite *increase in leukocytes*. *Leukocytes are very scarce in healthy bile. In diseased bile they are often definitely present.* Even in centrifugalized specimens of healthy bile only an occasional leukocyte is seen. By high power of the microscope bacteria can be readily distinguished in diseased bile. Smears of the centrifugalized bile can be made and studied. I feel that this method is worthy of study and should be used in all cases. Certain clinicians have called attention to the *turbid*



*bile* seen in certain cases of cholecystitis and angiocholitis. I have seen turbid bile on many occasions, but the turbidity was by no means always due to infection. In fact, there are certain forms of gastric juice which induce turbidity of the bile, and again, there are certain forms of bile which readily become turbid owing to chemical changes which occur. The difference in the *color* of the specimens have caused many clinicians to ask whether the bile was diseased. I have examined bile from many sources—angiocholitis, the various liver diseases, hemolytic jaundice in which bile appears—and I have seen every shade from a red brown to a pale straw color. This alteration is undoubtedly due to an alteration of pigment on the part of the liver cell and should form the basis of interesting studies in the future. One point is rather important, namely, the differential diagnosis of cancer of the head of the pancreas from chronic stone obstruction. It is claimed that in the former the obstruction is complete, while in the latter traces of bile can still be detected. I have been able to examine examples of both these conditions and have observed the total obstruction produced by cancer.

*Chemical Examination.*—Examination of bile should be possible from a chemical angle particularly in regard to: (a) pigment concentration, (b) the presence of urobilinogen and urobilin, (c) the concentration of bile salts, and (d) the concentration of cholesterol. These points are still under discussion, and practical methods have as yet not been satisfactorily evolved. In fact, the bile, as representing the external secretion of the liver, should offer a convenient method for the determination of liver function. The tetrachlorophthalein test is one of those in which the specific dye stuff is eliminated by the bile.

*It must be remembered that in the direct examination of the bile by duodenal intubation the possibility of material being duodenal or pancreatic should be constantly entertained.*

The presence of bile pigments and acids in other body fluids than bile must be considered, inasmuch as their finding is often of great importance. In obstructive jaundice the bile pigments and acids may be found in the blood, urine, and even in the sputum and certain exudates. Its recognition in these various

fluids is of importance. It is claimed that normally the pigments and acids are not found in the blood, but Croftan has shown that bile acids may occur or be found in the blood of healthy persons, probably accounted for by the fact that bile pigments are reabsorbed and re-excreted in the bile (Webster, *Diagnostic Methods*, 5th ed., p. 451, Weintraud—Webster).

*Tests for Bile Pigments.*—Practically all these tests depend on the oxidation of bilirubin into the colored derivatives, *e. g.*, biliverdin—green, bilicyanin—blue, choletelin—yellow.

(b) *Indirect Analysis of Bile.*—Fecal analysis will reveal certain syndromes which correspond with: (1) deficiency in bile, which may be either obstructive or due to the lack of formation of bile, in which there is no systemic absorption, and (2) biliary hypersecretion or polycholia.

*Hypocholia.*—The characteristics of the feces when there is diminution of bile or *biliary insufficiency* are as follows (Gaultier):

(1) The duration of digestion is prolonged usually in direct proportion to the shut-down of bile.

(2) The relationship of the weight of the dried feces to the moist fresh feces is altered, there being a diminution of water with an increase in the total dried feces.

(3) The reaction of the feces is acid.

(4) Fats are distinctly diminished from the standpoint of utilization, more than one-third being excreted, and in a different form from that seen in the normal stool. The neutral fats represent more than one-half the total fats in contrast with the split fats, such as the fatty acids and soaps.

(5) The color of the movement is altered in the direction of acholia, and there is a definite diminution in the amount of bile pigment as measured by Gmelin's reagent.

*Hypercholia*, or *biliary hypersecretion*, is shown by the following facts:

(1) There is a shortening in the time of digestion (total transit time).

(2) There is an increase in the amount of water with a diminution of the amount of dried substances.

(3) The reaction of the feces is acid.

(4) There is a general increase in the various alimentary substances which are not transformed (often due to rapid transit), and a marked increase in the amount of bile pigment, easily demonstrated by Gmelin's reagent.

This syndrome must be distinguished from (1) the pure pancreatic deficiency, and (2) the associated biliary and pancreatic syndrome into which the *biliary insufficiency is liable to merge*.

*Pancreatic insufficiency* is associated with a short digestive transit, a general decrease in the digestibility of all the food-stuffs—proteins, fats, and carbohydrates—a neutral or alkaline reaction due to putrefaction of unabsorbed albumins, while the quantity of unabsorbed fats is very markedly lowered, more than two-thirds of the undigested fats being neutral and not split. Every variation of biliary and pancreatic deficiency can be encountered. These facts can be demonstrated macroscopically, microscopally, and by chemical analysis.

*Intermittent Elimination of Bile.*—This is an important point to be borne in mind, and is characteristic of many of the functional disturbances of the upper gastro-intestinal tract. Intermittent output of bile may be seen in the same movement, part of it being light in color, the other portion well colored with bile. At times this apparent change is due to the conversion of bilirubin into the colorless hydrobilirubin, and therefore in every case both parts of the movement must be submitted to the action of Gmelin's reagent. On the other hand, in many instances the two parts of the movement show a totally different bile output. This may be accounted for in the dietary, there being at one meal substances which produce little bile flow, and at another substances causing a large emission of bile. However, in the normal movement there should not be this sharp deviation, and certainly not after the test diet, in which there are substances calculated to induce sufficient bile flow. In many of the forms of cholelithiasis, in hepatic insufficiency, and in various forms of duodenitis there is this intermittent flow. The most typical type would be a ball-valve stone in the ampulla, but here the type of movement is very different, it almost always being an acholia,

in which at intervals the trace of bile present becomes more pronounced. In my estimation *intermittent bile flow in the absence of evidence of systemic absorption of bile (jaundice bile in the urine, etc.) indicates a disturbance in liver function. Never depend entirely on the color of the movement; this should always be checked up by a test for the pigments.*

*Chemical Examination of the Feces for Bile.*—The point, however, which must be insisted upon is the necessity for determining the time interval in which the feces give the bile reaction. In some cases of obstructive jaundice the movement must be left in the incubator several hours before a positive reaction occurs with the concentrated corrosive sublimate solution. The study of the time required for the development of a positive reaction with biliary tests is a matter not only of clinical interest but also of extreme importance, inasmuch as there are many cases *in which there is a quantitative diminution of bile output without a disappearance.*

In every case of biliary and hepatic disease the questions which must be solved are the following:

(1) Is there obstruction to bile excretion implying a mechanical block? The syndrome of obstruction would be jaundice, bile in the urine or blood, and jaundice, with a disappearance of bile pigment in the feces (acholia).

(2) *Is there reduction in bile formation without obstruction?* This means a reduction of bile formation on the part of the liver. Obstruction, however, may occur in the higher biliary radicles in the liver, and not necessarily in the ducts, but a positive diminution in the formation of bile would be found to coincide with (a) a reduction of bile in the feces, (b) no signs of systemic absorption of bile.

(3) *Is there a hemolytic jaundice?* This would be accompanied by signs of hemolysis, by the obvious excretion of bile in the intestines and its presence in the feces, and the evidence of the peculiar type of jaundice seen in that condition; in other words, jaundice without acholia. I have on several occasions found the excretion of bile in, if anything, increased pigment concentration in hemolytic jaundice. Not only are the stools examined

for their bile content but also for fat digestion which follows the rules set forth above.

**2. Finally, there is Another Method by which the Gall-bladder Can Be Examined, and that is By Means of the x-Ray.**—Certain observers have claimed that as high as 50 per cent. of stones can be discovered by this means. This figure, in my estimation, is entirely too high, and unless the technic is very markedly improved, the physician cannot rely on the demonstration of more than 20 per cent. under the most expert hands. Briefly, the stones most likely to be detected are those in which there is a deposit of calcium salts. Pure cholesterol stones approximate so closely the specific gravity of the body tissues that their detection is difficult. Ordinarily we get a positive shadow, but Cole has described a method by which even negative shadows are given by the x-ray. This method can likewise, in not a few instances, reveal the size of the gall-bladder. *It is far more useful, however, for the detection of deforming lesions incident, for example, to a pericholecystitis.*

Before leaving completely methods of technic it is well to discuss a method of blood analysis which within recent years has given rise to considerable interest, namely, the estimation of *blood cholesterol*. In our laboratories we have examined many cases, in some of which stones were demonstrated at operation, and again many cases in which lesions other than those of the gall-bladder were found at operation. The principle of the test is that in health 160 to 180 mg. of cholesterol are normally found in 100 c.c. of blood-serum. In disease, owing to the processes inducing hypercholesterolemia, there is an increase over these figures of 200 to 800 mg. or more. The mechanism by which cholesterol is increased has already been mentioned under the heading Cholelithiasis, and the diagnostic interpretations to be drawn must always consider the fact that diseases other than cholelithiasis are accompanied by hypercholesterolemia. In our experience about 70 per cent. of operatively demonstrable gall-stone cases reveal this finding, while in ulcer of the stomach and duodenum its presence is rare. In about 30 per cent. of cases no increase is found, due either to the fact that the stones

are not cholesterol stones (low in cholesterol) or to the fact that this particular mechanism, so active while stone was formed, has ceased after the developemnt of the stone. Further research is necessary before the exact status of this method is settled, but its determination is often of value as furnishing one more link in the chain of evidence for or against stone.

*Determination of Blood Cholesterol.*—Method of Autenreith and Funk. This is the method used in our laboratory, and according to Hawk is performed as follows:

*Principle.*—The blood-serum is boiled with strong alkali to saponify the fats. The alkaline solution is extracted with chloroform. The chloroform is then dried and clarified by means of anhydrous sodium sulphate and filtration and treated with sulphuric acid and acetic anhydrid to develop the characteristic color reaction (Liebermann, Buchard). The color is compared with a standard in the colorimeter.

*Method.*—With an accurate pipet transfer 2 c.c. of blood-serum (collected with an intravenous needle from a vein in the arm) to a 100 c.c. Erlenmeyer flask, and 20 c.c. of a 25 per cent. potassium hydroxid solution is added: Heat on the water-bath (with a reflux condenser apparatus attached) for two hours, shaking frequently, and adding a little water if necessary to prevent the solution evaporating to dryness. Pour the undiluted mixture into a separatory funnel and add 25 to 30 c.c. of chloroform. Shake vigorously for five minutes and separate. Shake out four more portions with 20 c.c. of chloroform each. The combined chloroform extracts are turbid and of a green or brown color, but are clarified by adding 5 to 10 grams of anhydrous sodium sulphate and shaking, then filtering. Dilute the filtrate to 100 c.c. with more chloroform. Transfer 5 c.c. of this extract to a small glass-stoppered bottle of about 10 c.c. capacity, add 2 c.c. of acetic anhydrid, and 0.1 c.c. of a concentrated sulphuric acid, and shake. Place in a water-bath at 32° to 35° C. and keep in the dark about fifteen minutes. A green color is developed. At the same time a series of standards are prepared and treated in the same manner. The color of the test is compared with the most similar standard in a colorimeter.

Five standards are kept, these being prepared by dissolving in 100 c.c. portions of chloroform: (1) 3.2; (2) 4.8; (3) 6.4; (4) 8.0; (5) 9.6 mg. respectively of pure cholesterol. In preparing the standards for comparison, 5 c.c. portions of each of the above solutions are taken, placed in glass-stoppered bottles, and treated as were the unknowns. Each standard then represents a concentration of: 160 mg.; 240 mg.; 320 mg.; 400 mg.; 480 mg. respectively of cholesterol in 100 c.c. of the original blood or serum. We use the Duboscq colorimeter, in which the amounts of the colored substance are inversely proportional to the depths of the column of liquid.

**Correlative Diagnosis.**—In reaching a diagnosis of gall-bladder disease in almost every instance a correlative diagnosis must be made by carefully reviewing all the evidence—etiologic, symptomatic, physical, and chemical—and comparing all these. Many clinicians depend on a careful history, and a careful history cannot be overestimated, inasmuch as the character of gall-bladder attacks, while in certain cases absolutely typical, can be most atypical. In those cases where physical examination reveals findings definitely located in the gall-bladder area, and there is associated typical attacks of colic, the difficulties in diagnosis vanish, but in those cases of obscure, chronic gastro-intestinal ailments of long duration the differential diagnosis can be most difficult.

*In Every Case of Chronic Gastro-intestinal Disease, in which There is a Suspicion of Gall-bladder Trouble, Each of the Methods Described Must Be Investigated.*—The presence of tenderness in the gall-bladder area, the presence of hyperesthesia over this region, the presence of atypical rather than periodic attacks, the presence of an intermittent bile flow in the feces, the presence of an aberration in fat absorption alone, the presence of traces of bile in the blood-serum or urine, each or any one of these symptoms *should suggest a careful examination of the gall-bladder area.* No examination of the gall-bladder area is complete which does not have also an examination of the bile, because *the bile is the only visible evidence of function which we can obtain.* *Aërophagia, flatulence, gastric distention, anorexia, heart-burn, nausea, or*



*vomiting while symptoms of any gastro-intestinal disease, nevertheless occur with such frequency that they should give rise to the suspicion of gall-bladder disease.*

An antecedent history of cholangitis or angiocholitis should always excite one's suspicion of cholecystitis. Evidence of duodenitis should always suggest the possibility of angiocholitis and cholecystitis. Furthermore, the clinician must always have in mind the varied etiology of chronic and acute infections of the gall-bladder, and be prepared, first, to accept the fact that in many instances gall-bladder disease is secondary to a primary focus elsewhere in the body, and second, to remember that the association of gall-bladder disease with other organs, particularly the appendix, is exceedingly common. A diagnosis of cholecystitis or cholelithiasis does not rule out chronic appendicitis, and vice versa. *Another point well worthy of mention is the association of gall-bladder disease with evidence of aberrant liver function.* In fact, Chauffard has repeatedly insisted upon the necessity of considering the primary cause of cholelithiasis as in the liver cell, where bile is formed, and not in the gall-bladder, a thought which is substantiated by many of the phenomena of cholelithiasis. With deforming lesions like pericholecystitis the problem is different, and the deformity produced in adjacent organs (duodenum and pylorus) may determine the clinical picture as due to disease of those organs.



## CLINIC OF DR. B. B. VINCENT LYON

JEFFERSON HOSPITAL

---

### SOME ASPECTS OF THE DIAGNOSIS AND TREATMENT OF CHOLECYSTITIS AND CHOLELITHIASIS

#### DISCUSSION OF A NEW METHOD

GENTLEMEN:

I wish to bring to your attention today the very interesting case of the gentleman who sits before you, and to whom I shall refer as Mr. J. H.

You will notice that he looks like a pretty healthy specimen of manhood. His age is forty-seven, his present weight is 182 pounds. His color is excellent, his eye is clear, and his tongue is clean. Three months ago he was a very differently appearing man. He was diagnosed as a case of cholelithiasis, cholecystitis, and choledochitis. He has been operated upon for the removal of gall-stones with surgical gall-bladder drainage, followed by drainage of the biliary system (including the ducts, gall-bladder, and liver) by a new and non-surgical method that I especially desire to bring to your notice because it opens up for us an entirely new field of usefulness. This non-surgical method of biliary drainage can be utilized for both diagnosis and treatment.

The diagnostic problem that this gentleman presented was somewhat complex. But let me begin at the very beginning:

This Mr. J. H. was referred to me by Dr. Orton, of the Pennsylvania Hospital. He arrived at my office on September 29, 1919, and gave me the following history: He had always enjoyed truly robust health until he contracted influenza in October, 1918, just one year ago. This was of the so-called "Spanish flu" variety common to the pandemic which was then raging. He was quite sick for eight days in bed, and I will ask you to remember that he had associated with the influenza

symptoms a catarrhal rhinitis and catarrhal bronchitis with copious secretion. He states that this attack of influenza weakened him greatly, that his convalescence was slow, and that he has never really felt well since. Questioned as to other infections in the past, he told me that he had had none—indeed, that he had never been sick in bed from a genuine medical illness during his adult life of twenty-five years.

On cross-questioning, however, I learned that for nearly twenty-five years he had had attacks of headache, occurring about once a month and lasting for two or three days—blinding headaches with dancing black specks before his eyes. They progressed from occipital to temporal, to frontal in location, and when most severe were accompanied by dizziness and even vertigo, with occasionally actual falls. At these times he found he had to go to bed for a day. At the height of the attack he would break out in chilly cold sweats and would occasionally have vomiting—quite often in the beginning of the biliary type. These attacks were only relieved by calomel and salts, and later the severity of the attacks was partially controlled by wearing glasses. But their frequency and their cyclic recurrence was not altered. He also stated the interesting fact that his headache attacks were always preceded by intense hunger, that in the beginning he would gratify this desire to eat, and overeat, but soon learned that it made his attack much worse.

Now, gentlemen, I will ask you to mentally pigeonhole this phase of his case. For you will find it useful in the future to realize that these cyclic attacks of headache, associated with dizziness, lethargy, nausea, and vomiting and relieved by calomel or other of the so-called cholegogic groups of medicines, are of very common incidence and unhappily passed over too indifferently by many of us. They represent conditions variously described as "hepatic torpor," "biliousness," "sluggish or lazy liver," "migraine," etc., but in the end-analysis it means biliary stasis of some degree from simple sluggish secretion, with sluggish flow, to actual partial obstruction due to slight catarrhal swelling in the ducts, and here we have the added symptom or objective finding of jaundice ranging from the slightest icteroidal

tinging to even what might be designated a mild catarrhal jaundice.

And here let me digress this much to state my personal belief (and, indeed, this may prove to be prophetic), that if we are to attack the great problem of gall-bladder disease, gall-stones and gall-bladder and duct catarrhs and infections, and attack it at its source, we must give this lightly passed over symptom-grouping called "biliousness" our serious attention. Thus far our attitude toward the gall-bladder problem has been one of *correction* of the full-blown stages of formed calculi and active catarrhal infection, and the means adopted have been largely surgical. Their results have been at times brilliant, often less satisfactory, and quite frequently bad, requiring many surgical maneuvers, usually eventuating in that *bête noir* of surgery, distorting postoperative or postinflammatory adhesions, so that the state of chronic invalidism of the patient is a difficult cross to bear. What we must do is to attack the problem with methods for *prevention* of gall-bladder disease with its sequelæ, and this brings us back to attacking biliary stasis which is at the root of the matter. Biliary stasis is followed by overdistention of gall-bladder and ducts, leading perhaps to what we may designate in the future as gall-bladder atony—this engenders catarrhal states of gall-bladder and duct mucosa, weakening resistance, and permits of successful implantation of infecting micro-organisms, filtered out from the portal blood by the liver, or carried directly to the gall-bladder by the systemic blood or by the lymphatics, or ascending to the gall-bladder by way of the duodenum and the common duct. Biliary stasis with its concentrated bile and precipitation of its crystalline chemistry plus catarrh plus infection means gall-stones. Therefore it is biliary stasis that we must attack if we are to prevent gall-stones, catarrhs, or infections. Can we do so? Possibly we can if we keep driving at the problem by the method that I am rather sketchily going to bring to your attention during this hour.

Now let me return to a consideration of the present illness of Mr. J. H.: Four months ago, or eight months after his attack of influenza, he began to have sudden attacks of epigastric dis-

tress ushered in by nausea, followed by pain at first of a "smothery" type, later cramp-like, but not referred to back or to either shoulder. Still later the pain became a steady ache, the nausea increased, and one to two hours after eating he would vomit a sour but not bitter vomitus. On only one occasion had he noted retention vomiting, and this was of overnight or twelve hours' duration. Toward the end of the attack, which would last from a few hours to a day, he would become very mildly jaundiced for a few days and have somewhat diffuse epigastric soreness. His stools were offensive and "gassy"—floating on the toilet water—therefore fermentative, and were a light colored yellow. He had lost 31 pounds in four months. During this time he has had five attacks, the last one being ten days ago.

On physical examination I found him a man of about 5 feet, 11 inches, of large frame, and weighing 161 pounds. He showed his loss of weight and looked flabby. He was visibly anemic and had a worried expression. His teeth were in bad shape and a number had been extracted. He undoubtedly has had oral sepsis. His tongue was clean. The blood-pressure of 120-86 was low for his years. He had no glandular enlargements. His lungs and heart were excellent for his years except that the aortic second sound was "low."

His abdomen, except for being flabby and somewhat fat and the presence of several petechiæ and pigmented warts, was negative. He had absolutely no tenderness or rigidity of muscles; especially were the three important points—duodenal, gall-bladder, and appendix—negative for tenderness or sensitivity. Nor had he any tender points down his spinal column. His pupils, reflexes, deep reflexes, and equilibrium tests were normal. He had a right inguinal hernia, for which he was wearing a truss. Rectal examination revealed nothing abnormal except slight internal hemorrhoids.

During examination he was nervous, fearful, and apparently holding himself under tension.

**Special Examinations.**—His urine contained only slight traces of albumin. The "P. S. P." test showed 68 per cent. of elimination in two hours' time. His blood showed a moderate

secondary anemia and a normal leukocyte count. The Wassermann reaction was sharply negative.

Thus far we have very little positive laboratory help in diagnosis. We find more assistance in examination of his gastro-intestinal tract.

A twelve-hour motor meal shows no gross or microscopic retention. The twelve-hour fasting residuum amounted to 25 mls., very syrupy, and a golden yellow, therefore biliary regurgitation. It is quite turbid, with flocculogranular precipitate microscopically containing many large pigmented so-called heart failure cells (no longer to be called "herz fehlen zellen"), and please pigeonhole this with the aforesaid note of influenza with catarrhal bronchitis; many pus-cells with protoplasm intact; many cocci and bacilli; no sarcinae; many bile crystals; much endogenous and some exogenous epithelium. Chemically there was no Free HCl and the total acidity was 15 acidity per cent., and there was an instantaneous blood reaction to benzidene. Lactic acid was demonstrable.

The fractional analysis of a bread-and-water meal showed nearly complete achylia (free hydrochloric acid to 10 acidity per cent. appearing at one and one-quarter hours, and total acidity ranging from ten to twenty-five); hypermotility; biliary regurgitation in four specimens, traces of bleeding in all eight increasing as the stomach becomes empty; and a steadily increasing amount of mucus in each specimen. The microscopy of several specimens showed mucopus and bacteria. These examinations opened up interesting possibilities. "Frank" fasting stomach biliary regurgitation is a very significant thing, although I am well aware that several reliable authorities say that they encounter it in nearly 50 per cent. of their normal patients. To my mind it is important and makes me alert to chiefly extragastric possibilities (particularly gall-bladder, duodenal, or appendical), for the physiologic mechanism of the biliary tract is disturbed either in itself or reflexly.

Digesting stomach biliary regurgitation, especially if the acidity curve is low, strengthens my feeling of organic disease whether or not regurgitant pus or blood can be found chiefly

in such specimens (of course in the latter case it makes a duodenal lesion, duodenitis, or ulcer almost sure). Watch this in the future and see how often you are dealing with gall-bladder or duct disease, duodenal ulcer with adhesions, or chronic appendicitis of the adhesive or obliterative types with often an incompetent ileocecal valve. Alvarez's studies of reversed peristalsis have strengthened my view.

Analyzing our facts so far, the two chief possibilities in diagnosis appear to me to be gall-bladder disease or carcinoma of the stomach or its neighborhood. In favor of carcinoma so far in the findings we have 31 pounds loss of weight, progressive anemia, pus and blood in the fasting and digesting stomach, and the presence of rod-bacilli, suggesting the Oppler-Boas and the presence of lactic acid. But on further examination we find that the Wolff-Junghans test for soluble albumin is low and not suggestive of cancer; the rod-like bacilli turn out by Gram's stain to be the *Leptothrix buccalis*; the pus and blood may mean an infected atrophic and congested mucosa, and suggests a preceding oral sepsis, and the blood-picture could as easily be from the toxemia of a chronic infection as from the continual small seepage of blood.

Stool examination, too, supports my feeling of gall-bladder disease in showing faulty fat digestion and insufficient bile-pigments, although we must consider pancreatic conditions somewhat closely. But it is when we come to the duodenobiliary *direct analysis* that we find our diagnosis clinched.

This examination is done, of course, with the duodenal tube and on the fasting stomach, and the procedure is somewhat as follows:

Disinfect the mouth, assist the patient to swallow the tube, wash thoroughly and disinfect the stomach, then allow the marked tube to be swallowed *slowly* until it passes about 3 inches into the duodenum. Try to gently aspirate the duodenal secretion and set it aside for examination for catarrhal, inflammatory, or infective elements. Note whether the duodenum is bile free, indicating that the biliary sphincter is contracted, which it normally (with some exceptions) should be in the fast-

ing stomach and duodenal state. First disinfect and then locally douche the duodenum with 50 to 100 mls. of 25 per cent. saturated solution of magnesium sulphate, which serves to relax the tonus of the duodenum and of the circular muscle-fibers (Oddi's muscle) at the terminal end of the common bile-duct, connect up with the first aspirating bottle and note how soon bile begins to flow; continue to collect the bile in this bottle until through the glass window in the duodenal tube you notice a deepening in the color of bile, indicating that the first bile diluted with the magnesium sulphate solution is being replaced by pure bile; now disconnect the first bottle, set it aside for examination, attach bottle No. 2, and continue your collection of bile until it, too, changes in color and consistency, usually to a deeper yellow and thicker. Detach and lay aside and attach bottle No. 3, and continue drainage until this darker bile is replaced by a lighter lemon-yellow, limpid, and usually transparent bile, when you promptly detach your third bottle and attach the fourth, in which you now collect as many mls. as you care to. By this means you can keep on draining the biliary system for as many minutes or hours as you wish. All tubes, bottles, syringes, and glass receptacles used in this work are, of course, sterile.

The "A" bile in the first bottle must of necessity be the first bile available, or that in the common duct diluted with the magnesium sulphate solution; the "A" or second bottle of bile must be common duct bile undiluted with magnesium sulphate, but perhaps augmented slightly by gall-bladder bile and possibly by drippings from the right and left hepatic ducts; the "B" or third bottle bile will be found to be nearly pure gall-bladder bile, and is much larger in amount and more concentrated in color and consistency than either the "A" or "C" biles. The "C" bile, or that in the fourth bottle, is freshly secreted bile from the liver capillaries, to the best of my belief, because when this stage of limpid lemon-yellow bile is reached it rarely changes its color or consistency thereafter as long as you continue drainage, except in rather infrequent instances in which the gall-bladder has failed to completely empty its contents.



So now you will see that we have collected and segregated in different sterile bottles bile from the several components of the biliary system, the common duct (and cystic duct?), the gall-bladder, and from the liver itself. The contents of each bottle, together with the material first aspirated from the duodenum, can be *separately* studied microscopically, culturally, and chemically for pus, mucopus, epithelium, blood, crystals, etc. You will at once see the aid this gives for a direct differential diagnosis of diseases of the biliary system.

The whys and wherefores of what I have briefly outlined I will give to you in a subsequent lecture and will demonstrate the method on a selected patient for your benefit. For those who are interested in "reading up" on this subject I can refer you to my first published paper on this method which appeared in the Jour. Amer. Med. Assoc., September 27, 1919.

Applying this method of diagnosis to Mr. J. H. on October 3, 1919 I found that the tube reached the bile-free duodenum in twenty-two minutes; that the duodenal secretion showed evidence of a catarrhal duodenitis; after douching the duodenum with magnesium sulphate solution bile appeared in six minutes (somewhat delayed). The "A" bile measured 30 mls., was a light golden yellow, syrupy, and nearly transparent bile, with a few floccules of mucus and microscopically contained bacteria. The "B" bile, measuring 85 mls., was a deep golden yellow, very viscid and quite distinctly turbid, and contained little bright yellow flecks that settled at the bottom. Microscopically it contained pus and mucopus, running perhaps 100 polys. to the microscopic field, bile-stained columnar epithelium, and swarms of bacteria. The yellow flecks were enormous clusters of bile crystals. After culturing the bile I poured some out in a plate and found that where the yellow flecks lay they gave a sense of grittiness, like sand, to the finger.

The cultures were sent to Dr. Sappington, who later reported the following bacterial identifications:

*B. coli* communior; *Pneumococcus capsulatus*; *Streptococcus viridans*, and *Micrococcus tetragenus*.

Here, then, was a solution to our diagnostic problem worked



out in a scientifically correct manner by a direct method. It was now easy to say that this patient had an infected duodenitis, a cholecystitis, and a choledochitis, with, most probably, gall-stones. The latter was suggested by the free deposition of biliary crystals giving a grittiness to the bile from the gall-bladder.

Let me now direct your attention again to the points I have asked you to mentally pigeonhole. You will remember that this patient had "pandemic" influenza, with an associated catarrhal bronchitis and rhinitis. This inevitably means the swallowing into the stomach of infective material. One year later I am able to recover from the stomach the so-called "heart-failure cells" so frequently seen in certain types of catarrhal bronchitis, especially of the capillary variety, together with other evidence of an infective gastritis. Incidentally, I have demonstrated a specific infective gastritis in a great many post-influenzal cases during the past year. You will remember, too, the finding in this case of an infective duodenitis. To make my point more clear I am sorry now that I did not attempt to obtain direct cultural identification from the stomach and duodenum. But consider now the bacteriology of the common duct and gall-bladder biles. Does not the recovery from the bile of the pneumococcus, the streptococcus, and the Micrococcus tetragenus, three organisms so commonly seen in the recent pandemic of influenza, have a special significance in connecting up cause and effect? Although it is quite possible for this group of organisms to have reached the gall-bladder by way of the systemic blood, does it not seem more probable that the route in this case was by way of an infected stomach and duodenum, with ascending infection of the bile-duct and bladder?

Also I would have you recollect that this patient gave a history of many years of biliary stasis, followed by mild catarrhal states, and the two factors combined serve to lower local mucosal resistance. After catching the only acute infection in his life, this man within less than a year develops a cholecystitis with recoverable organisms, strikingly parallel to those seen in many influenza cases; and with this infection superimposed on stasis

and catarrh he had, I believe, a recent formation of gall-stones—why recent? Let me explain this later as I proceed with his case.

On October 8, 1919 Dr. Manges gave me the report of his x-ray examination, which, summarized, is as follows:

"Probable cholesterin stones in gall-bladder. No organic lesion of stomach or intestines. Kidneys, ureters, and bladder negative as to calculi. Chest normal to fluoroscopy."

On October 16, 1919 he was operated on by Dr. J. D. Elliott, who had ten years previously removed a lipoma from this patient's side. Through an upper right rectus incision the stomach, duodenum, and pancreas were examined and found surgically normal. On exposing the gall-bladder it was found shrunk and contracted, with thickened wall, and contained fifty to sixty small unfaceted stones ranging in size from small granules to that of a large match-head. The bile was thickened and of the color of that recovered in "B" bottle; cultures made by puncturing the unopened gall-bladder with a hypodermic needle were reported sterile. This seems to me a very poor way to obtain a bacteriologic proof from any gall-bladder and accounts for many discrepancies. By this needle method only the supernatant bile is recovered, and will give far less constant positive returns than cultures planted from material derived from the floor of the gall-bladder, and especially from the mucopurulent flakes that represent exfoliation from the gall-bladder wall.

Mr. J. H.'s gall-bladder was not removed, but was drained by rubber tubing sewed in with chromic gut; the total duration of drainage was eleven days; seven by tube and four days of drainage into the dressings. The total amount of bile drained by the rubber tube and recorded in bedside notes was a little less than 8 ounces in the seven days; the four days of dressing drainage could not be accurately measured, but probably did not exceed very greatly that recovered by the tube. The patient made an uninterrupted surgical recovery and was discharged at the end of his fourth week. Aside from the removal of his gall-stones, was this patient cured of his infection of the

gall-bladder? Leaving out the question of gall-stones, for they are by no means present in every case of cholecystitis or of microbic infection, undoubtedly some are simple inflammatory catarrhal states, so I say, that aside from the question of cholelithiasis the principal of surgical cures depends solely on the principle of free surgical drainage plus that of bed rest and arrangement of diet, to which surgeons pay too little attention. This you will see has natural limitations.

Dealing either with infected or simple catarrhal cholecystitis whether or not the patient is free entirely from his trouble will depend on how long drainage is continued and how free that drainage may be. It stands to reason that in any given case, for instance, Mr. J. H. of infected cholecystitis, where surgical drainage was conducted for eleven days, if the infection is not arrested by expiration of the time of surgical drainage his infection is not cured; it is, of course, better temporarily, but more frequently than the surgeon, I am sure, cares to see, the infection lights up again or the biliary stasis plus the catarrh gives rise to a redevelopment of gall-stones, and again requires surgical intervention. This time the gall-bladder may be removed and the common duct drained. But this does not always result in cure either, as is evidenced by the fact that I have had recently under my care one patient who was operated on four times in five years, and another three times in two years, and yet who continued to have intermittent attacks of acute colic with jaundice and whose common duct drainage still could be demonstrated as infected. By this time inflammatory and postsurgical adhesions complicate the situation, and may contraindicate further surgery, even from the surgical viewpoint, on the ground that it may make matters worse, and so these patients are destined to chronic invalidism unless some other remedy can be devised.

When simple gall-bladder drainage failed to give satisfactory cures, the explanation was furnished that the walls of the gall-bladder itself were diseased and infected, and logically, therefore, the gall-bladder should be removed as being the source of the infection, and hence the recent increase in the number of chole-

cystectomies, usually with drainage of the common duct. Improvement has followed this course of procedure so long as the common duct has been entirely freed of its infection. If not, the patient is in deeper water than ever, for deprived of the physiologic function of the gall-bladder as a distensible storage chamber for excess liver bile, to be delivered in concentrated form as needed in the digestion of certain essential forms of food, if now the infected common duct becomes partially or totally blocked the secreted liver bile must dam up between the closed sphincter and the liver, with the result first of dilatation of the common and hepatic ducts and later the development of biliary cirrhosis of the liver. Some surgeons have expressed the opinion that the gall-bladder is an organ non-essential to the continuance of life and that human beings can safely do without them. Granted that this is, in many instances, true, it is because the physiologic nervous mechanism of the biliary apparatus is fortunately broken when the gall-bladder is removed, and the sphincter of the common duct remains relaxed, and bile is continuously, rather than intermittently, poured into the duodenum. This has been my observation in postoperative studies on several cholecystectomized patients. Even if the common duct becomes partially or completely obstructed, it is possible that the ducts and liver can continue for a short time to vicariously assume the function of the gall-bladder, but sooner or later this too breaks down.

In support of their contention that the gall-bladder does not possess a physiologic function necessary to continuance of life and good health, surgeons point to the fact that the deer and the elk manage to get along excellently well without a gall-bladder. Such analogy is instantly open to attack, because the animals referred to are strictly herbivorous and need not be supplied with a reservoir of bile to be called upon to meet the demands of digestion of animal proteins, albumoses, and fats. Man is by nature and usually by choice a carnivorous animal, and if we search the carnivora, although my knowledge on this point may be incomplete, I think we will fail to find one which is without a gall-bladder, save, perhaps, a freak of nature.

The solution of the problem goes deeper than our present acceptable methods of treatment. As an aid to solving this I have taken up the postoperative study and treatment of the biliary tract by the method I have referred to earlier as diagnostically useful.

I applied this in the case of Mr. J. H. to see whether or not the surgical drainage of his gall-bladder of eleven days' duration had served to cure his infection. He returned to me for re-examination on November 27, 1919, and thereafter at weekly intervals. The method of procedure has been to disinfect his mouth thoroughly, pass the tube, and thoroughly wash and disinfect his stomach, allow the tube to pass to the duodenum, which, in turn, is rinsed and disinfected, and then, by means of the harmonic (?) action of magnesium sulphate, the biliary sphincter is relaxed and the biliary system is drained of all the bile that I can get until the clear lemon-yellow, limpid bile is being alone recovered, when drainage for the day is discontinued; the duodenum is again disinfected and a transduodenal lavage is given of Ringer's solution, boric acid, or normal saline. Autogenous vaccines from the bile-infecting organisms will be later given.

Of course all biles are segregated into the several sterile bottles for special cytologic and bacteriologic study after each treatment, and thus it is possible to keep a *direct check* on the *actual efficiency of treatment* to be balanced up with the symptomatic improvement.

I will now present for your final consideration the progress of Mr. J. H.'s case with the notes copied from my office record of the weekly findings in his case:

In each note it is understood that mouth and stomach cleansing and disinfection precede the entrance of the tube into the duodenum, and hereafter I shall allude to the "A" bile as meaning the "A" bile so far as the amounts are concerned, discarding the "A" bile mixed with magnesium sulphate solution, except as it is studied cytologically and bacteriologically. I wish it understood, too, that in the "A" bile or common duct bile there must be mingled *some* "B" or gall-bladder bile and a few drops

of "C" or liver bile, for rarely will it be able to segregate bile, on color estimation alone, as sharply as that; and similarly, in "B" or gall-bladder bile there will be an intermixture of a few drops of "C" or liver bile, depending, perhaps, on how slowly or rapidly the gall-bladder empties itself.

On November 27, 1919, or six weeks after the day of operation, the first examination was made, with these notes of findings:

Tube to duodenum: Bile free (the gall-bladder not being removed the physiologic double-nerve mechanism is not broken, and bile is, as a rule, being intermittently rather than continuously excreted into the intestines. This I think to be the normal physiologic mechanism, although this may be broken, too, where the gall-bladder apparatus is nervously or reflexly irritated. I find that when the gall-bladder has been removed the common duct sphincter *remains* relaxed and that bile is being continuously discharged into the duodenum, and that where it is not you can look for catarrhal, inflammatory, or other obstruction in the common duct).

Magnesium sulphate douche: 25 per cent.—60 mls. Bile appeared in duodenum in seven minutes. "A" bile light golden yellow, clear, with few floccules—amount, 40 mls. "B" bile (first portion) deep golden yellow, viscid, nearly transparent except for few floccules. Second portion, representing terminal portion of gall-bladder bile (from floor of gall-bladder?), was very turbid, thicker, and contains numerous floccules of mucopurulent type, gradually sinking to bottom of bottle, and considerable stringy mucus. Total "B" bile, 94 mls. Microscopy of mucopurulent floccules: many degenerated pus cells—many heavily bile-stained columnar epithelial cells, cytologically that of gall-bladder. Many bacteria, bacilli (motile), and cocci easily differentiated and occurring in masses. Very few bile crystals. Inflammatory debris. Cultures sent to Dr. J. A. Kolmer, who reports on December 2, 1919: "B. coli, and an unidentified coccus which occurs as a diplococcus and is now beginning to grow as a staphylococcus."

December 2, 1919: Tube to duodenum entered in twenty-five minutes. Bile flowing without magnesium sulphate douche,

therefore common duct is relaxed. "A" bile light amber, clear, with very slight flocculogranular sediment. Amount, 20 mls. Duodenum rinsed with 80 mls. 25 per cent. magnesium sulphate (to encourage gall-bladder to contract), and in a very few minutes "B" bile appeared—much like the week previous, at first clear, golden yellow, then turbid and thicker, and with many mucopurulent floccules. Approximately 5 ounces (150 mls.) of this bile was recovered before "C" bile appeared, a light lemon yellow and clear and transparent. Microscopy of "B" bile: Very many pus-cells, running over 100 to the field—some of them degenerated. Many cocci and some motile bacilli.

*December 11, 1919:* Same procedure. Findings about the same, except that "B" bile measured 94 mls. and pus-cells appeared in state of greater disintegration. Inflammatory débris. Cultures planted in my office in bouillon flasks, blood-sugar, and plain agar, and sent immediately to Dr. Sappington.

*December 15, 1919:* Dr. Sappington reports: "B. coli communior; Pneumococcus capsulatus; Streptococcus viridans, Micrococcus tetragenus."

This I considered extremely interesting and important because it represents identically the same group of organisms isolated originally from this man's bile, and recoverable again eight weeks after his operation. This, in association with pathologic bile containing pus-cells and inflammatory débris, is extremely suggestive, and affords the nearest direct scientific proof at present available that the length of surgical drainage in this case (eleven days) was not sufficient to rid him of his infection.

It is quite possible, too, that the organism growing as a diplococcus in Dr. Kolmer's cultures may have been the pneumococcus and have been overgrown.

*December 18, 1919:* Same procedure. "A" bile same in appearance—32 mls. "B" bile 40 mls., of very much better appearance, with less mucopurulent floccules. Microscopy of "B" bile: Fewer pus-cells and show more disintegration. Bacterial flora still very evident—motile bacilli and cocci. Less inflammatory débris.

*December 26, 1919:* Tube to duodenum entered in twenty



minutes—bile flowing without douching with magnesium sulphate.

1st bottle: 60 mls., representing common duct and gall-bladder bile.

2d bottle: 91 mls., representing gall-bladder and liver bile.

In second bottle were found the larger quantity of mucopurulent flakes and bacteria as before—bile thinner—still fairly deep yellow, with slight greenish cast, but much improved over earlier observations, and less mucopus.

*January 2, 1920:* Tube to duodenum passed on second attempt. "A" bile 18 mls., transparent, clear, with very few floccules, light golden yellow.

"B" bile 40 mls., transparent, clear, with mucopurulent flakes less numerous than seen before. Color deep golden yellow and very mucoid.

Microscopy: Notably improved. Fewer pus-cells and bacteria not so easily demonstrable.

On adding dilute HCl to the clear golden yellow bile I get a turbidity varying in intensity with the strength of the acid. In microscopic slides from this I can identify lecithin(?) and neutral fats(?) and many small spheroids that have an active dancing movement and don't "take" soudan III. This I have often seen before in those patients who have a fairly high gastric juice, when a spurt of this passes the pylorus, enters the duodenum, and mixes with the bile. But in this specimen of bile I find for the first time that in addition to the turbidity and precipitate there was marked effervescence which continued for some time, suggesting the reaction between carbonates and acid. Query: Has this patient a calcium carbonate diathesis and were his gall-stones partly or wholly made up of that variety? Look into this further. Clinically he is doing splendidly; his hemoglobin is normal; color much improved, and he has gained 21 pounds in weight.

This, gentlemen, brings us up to the present. I will keep you informed of subsequent events in this case.

There are many other aspects of this method that I shall hope to bring to your notice later on.



## CLINIC OF DR. E. J. G. BEARDSLEY

JEFFERSON HOSPITAL

---

### CHRONIC VALVULAR HEART DISEASE

#### Aids in the Diagnosis; Demonstration of Patients

THE excuse, if an excuse is necessary, for devoting an entire clinic to a consideration of the prevalence of chronic endocarditis, its known etiologic factors, and a discussion of the physical signs of the disease found in the 2 patients to be exhibited who are unfortunate enough to have "crippled hearts" as a result of this disease, lies in the frequency with which patients similarly affected are encountered in the daily work of every general practitioner of medicine.

Organic heart affections comprise, as you cannot have failed to observe, a very considerable proportion of the disabilities of the patients who occupy beds in all the medical wards, while every medical dispensary has an extensive list of the names of cardiac patients who are under its supervision.

If one were to visualize a parade of motor trucks occupied by all the available victims of organic heart disease of any city or town one would be greatly shocked at the surprising and alarming numbers involved in such a melancholy spectacle. In such a gathering of patients would be infants and children, youths of both sexes, and the adolescents, men and women in the prime of life, and aged persons whose lives were nearly spent. Among such a group of patients would be found the representatives of all the social classes. The pauper would be seen as well as the rich man, the laborer and the captain of industry, the professional man and woman, and the man and woman who toiled with their hands. To witness such a tragic pageant, even by means of the

imagination, should deeply impress the members of the medical profession with the importance of adopting methods that will aid in preventing a disease that adds such a serious handicap to the life and to the chances of success and happiness of so many of our citizens.

It is a recognized principle of preventive medicine that in any serious attempt to control or lessen the incidence of an infectious disease it is necessary to acquire information concerning (1) the prevalence of the disease in the community, (2) its cause or causes, (3) how the disease may be early recognized, and how its severe forms and its complications may be prevented. That much excellent work has already been accomplished in investigating, both at the bedside and in the laboratory, various phases of the diseases that frequently cause involvement of the endocardium simply attracts our attention to the fact that we have yet to learn how endocarditis can be prevented. Until we learn definitely how this disease may be prevented it is our duty to see that our patients' hearts are particularly safeguarded, as far as our present knowledge renders this possible, from the insidious dangers of endocarditis during attacks of all the acute infections. Without further knowledge than we now possess we are able to state positively that to a certain extent endocarditis may be prevented by giving greater care and intensive study to such infections as acute and chronic tonsillitis, gonorrhea, scarlet fever, and measles. The period of convalescence will often be much lengthened, with great benefit to the patient, if instead of accepting the clinical thermometer to be the only guide, we make a careful examination of the circulatory apparatus. The necessity of such careful and systematic examinations is even more necessary in and following such illnesses as acute rheumatic fever, chorea, typhoid fever, pneumonia, syphilis, gonorrhea, and influenza. We will do much to prevent the crippling of hearts if we consistently urge the removal of foci of infections from various portions of the body. Whether it be infected tonsils and adenoids, sinus involvement, alveolar abscesses, empyemata, chronically infected appendices, and gall-bladders, we should use our influence to have such cases carefully

studied as to what proceeding is safest and best for the future of the patient.

That removing foci of infection potentially saves certain patients from cardiac involvement is certainly true, and those of us who see so many weary and worn patients spending their last days, as one of them expressed it, "in slipping off a bed rest," feel that the conservative surgeon removes the infected organs, while the radical surgeon sometimes advises against the removal of organs that are "under suspicion of being foci of infection" because there may not be overwhelming evidence against them.

To recognize an acute inflammation of the endocardium at a time when cure of the condition or at least great improvement can be reasonably expected one must anticipate by weeks, months, or years the physical signs and symptoms that are commonly and truly ascribed to the terminal stages of chronic endocarditis. Marked cardiac enlargement, the presence of thrills and murmurs usually indicate a gross lesion of considerable extent and that has existed for a long period of time. On the other hand, rapidity of the pulse, slight alterations of the heart sounds, both as to quality and rhythm, and slight changes in the size of the heart, such changes not being otherwise reasonably accounted for, are suggestive of the necessity of suspecting the presence of an early endocarditis or myocarditis, and of treating the patient as though such a condition were present. When one views the intricate mechanism of the heart and contemplates the amount of work that this organ performs under conditions of stress and strain and then considers the occasional floods of bacterial, chemical, and toxic abuse that this organ must resist it seems less strange that cardiac disease is prevalent than that so many normal hearts are met with in aged persons. In spite of the fact that the symptoms and signs of organic heart disease receive much attention in the lectures, clinics, and ward classes of the clinical years of the undergraduate medical curriculum, the average senior student has personally examined with thoroughness so few patients with diseased hearts and (it is a pity to add) has examined with thoroughness so few normal hearts that he dreads a practical examination upon a "heart case." That this

is true indicates a serious fault in our system of teaching the art of medicine, and does not reflect upon the mental capacity of the student body. The truth seems to be that there are so many important subjects that must, of apparant necessity, be included in the course of study for the junior and senior years that the student finds little time and less opportunity for the intimate and practical investigation of cardiac patients either in the wards or in the dispensaries.

That there exists an apparently inexhaustible supply of clinical material for cardiac diagnosis makes it seem the more unfortunate that the supply cannot be more fully utilized for the professional training of the undergraduate students.

The temptation for both teacher and student is to feel that the deficiencies of undergraduate training will be compensated for in some mysterious manner during the year's hospital internship. Those students (fortunately of recent years few in number) who do not have the advantage of an internship are, as the soldiers are wont to say, "out of luck," and this is even more true of their patients. That residence in a hospital with the opportunities that present themselves may, for the exceptional student and in the exceptional hospital, compensate for lack of intensive, intimate, and personal instruction in the undergraduate course is perfectly possible, but that such compensation occurs to the average student in the average hospital is to be seriously questioned.

The usual experience of the recent graduate who enters the hospital as an intern is that he is assumed to know far more of the practical branches of medicine than he actually has had the opportunity of learning. In his new life in the hospital he receives little help or advice from the senior interns, who are busy with their own duties and problems, and less help from the members of the visiting staff. The intern learns by his own mistakes and spends a portion of his valuable hospital year in learning, without aid, facts that, had there been sufficient time and opportunity, he could have learned to better advantage in his senior and junior years under trained teachers. As one of the unfortunate results of the lack of practical familiarity with cardiac

diagnosis there exists an impression, current not only among students but among practitioners as well, that the diagnosis and particularly the differential diagnosis of valvular heart disease is an extremely difficult matter, and is an art only to be acquired by (so designated) "specialists." As a result of this very mistaken impression one frequently hears the opinion expressed that it is entirely unnecessary for the general practitioner of medicine to have definite knowledge as to the valve or valves involved in cardiac disease as long as the fact was appreciated that the patient was suffering with "heart disease."

If there are students in the clinic today who feel that accurate diagnoses in cardiac cases are beyond the ability of the average student, I hope to convince them that they are in error in this regard. If there is any one present, also, who considers it a matter of unimportance whether the correct diagnosis of a cardiac disorder is made, I hope to interest him in the pleasures of accurate diagnosis as well as to point out the absolute necessity of definite knowledge of the valve or valves involved in order to insure proper management and treatment, as well as to aid in forming a more accurate estimate of the prognosis as to the life and health expectation in any individual case.

We must remember that the majority of cardiac patients never enter a hospital and never consult other than a general practitioner of medicine. If the patient is fortunate enough to have as his or her physician a well-trained and thorough physician the specialist and even the hospital will seldom be necessary as far as the welfare and comfort of the patient are concerned.

The greater number of the men in this clinic will become general practitioners of medicine, and even those who aspire to special fields of endeavor will, let us hope, for a time practice general medicine. How then to become well-trained physicians and how to remain well trained is the problem that interests all of us. It is self-evident that the more often a student comes in intimate relation with persons who are ill, the more practical will become the student's outlook upon the problems of medicine and the less important will seem text-books except as works of reference.

The time is fast approaching, and it cannot come too soon, when the hours that are now devoted to lectures during the senior and junior years of the medical course will be spent in practical work at the bedside of the patients or in taking the histories and examining as well as treating, under proper supervision, the patients in the various dispensaries. The information obtained from books will assume a new meaning and interest when applied in conjunction with the practical work with a particular patient.

It is my purpose to bring before you 2 patients who complain of symptoms commonly associated with organic heart disease, namely, dyspnea, palpitation, and discomfort in the region of the heart, epigastric and upper abdominal distress, and edema of the extremities.

I wish, using these patients as demonstration subjects, to consider with you in a brief way the essentials of physical examination necessary to correctly diagnosticate and differentiate valvular heart lesions, and to demonstrate that such diagnoses can be correctly and relatively easily made in all but the exceptional cases, if the examiner (whether he be an undergraduate or a practitioner of medicine) is willing to spend the time necessary to systematically and thoroughly carry out a simple and entirely practical routine examination of the affected organ and the associated viscera.

Before we have the first patient admitted it will be well for us to mentally review the etiologic factors that commonly bring about the crippling of heart muscle and valves.

Let us tabulate the important diseases that have the unenviable reputation of causing endocarditis:

Acute rheumatic fever,	Gonorrhea,	Streptococcus infections,
Chorea,	Syphilis,	Staphylococcus infections,
Tonsillitis	Smallpox,	Pneumococcus infections,
Scarlet fever,	Influenza,	Meningococcus infections,
Erysipelas,	Measles,	Colon bacilli,
Diphtheria,	Mumps,	Tubercle bacilli.
Pneumonia,	Meningitis,	

In addition to the above formidable list we have: Gout, renal

disease, and arteriosclerosis, which have a tendency to bring about those changes in the heart valves that result in deformity of the valve and consequent difficulty with the circulation.

When we visualize this array of etiologic factors in the production of chronic endocarditis can any of us doubt the great value of a carefully elicited history in cardiac disease? That a past medical history is not always necessary in order that a correct diagnosis of a heart affection can be made is quite true, but that the diagnosis in many mild and obscure cases of endocarditis depends very largely upon a carefully elicited history is equally true.

In considering the list of etiologic factors that we have tabulated above it is well to remember that although every disease mentioned sometimes causes endocarditis, that the two most common causes in any large series of cases are acute rheumatic fever and chorea.

When one contemplates the amount of suffering, mental as well as physical, that results from the partial or complete incapacity of the victim of heart disease, it seems strange that there are not national societies formed for the purpose of studying and preventing such dangerous diseases as acute rheumatic fever and chorea or, for that matter, any of the other diseases that are frequent factors in the development of chronic endocarditis.

One must not, however, ignore the possibility of other diseases than those commonly looked upon as having injurious influences upon the endocardium being potentially dangerous. If all patients were carefully studied over a period of years following attacks of illness, there is little doubt that new and at present unsuspected etiologic agents would be discovered.

One of the very real difficulties encountered in any attempt to aid students, either undergraduate or graduate, to a better understanding of the various physical signs encountered in organically diseased hearts is their lack of familiarity with the physical signs to be found in normal hearts. The most important factor in determining a correct diagnosis from physical signs alone lies in the familiarity of the examiner with normal physical signs and their physiologic variation. The examiner must pos-



sess a mental standard of normality regarding physical signs, and this can only be acquired by painstaking examination of many normal individuals of varying ages, sex, build, occupation, and state of health. The advantages to be derived from an opportunity of examining large numbers of normal individuals is never to be ignored. Such examinations at gymnasiums, athletic clubs, etc., etc., are the very best preliminary training for those who would perfect their skill in cardiac diagnosis. To show enthusiasm for a careful examination of a normal heart is usually the first step toward a proper understanding of the physical signs encountered in abnormal hearts.

If one lacks the advantage derived from frequent examinations of normal hearts, one cannot too soon begin to acquire that knowledge.

It is hardly necessary to remind my hearers that an examination of any patient—infant, child, or adult—for any medical or surgical condition whatever that does not include an examination of the heart cannot be said to be complete or satisfactory. Here then lies the opportunity of every true student of medicine to familiarize himself thoroughly with the normal physical signs in examining every patient that consults him. Casual examinations are valueless to the patient and harmful both to the physician's skill and reputation. A thoroughly conducted examination, on the other hand, is a comfort to the patient and to his family, adds materially to the physician's skill, judgment, and training, and adds distinctly to the good repute of the profession of medicine. Any considerable experience in the examination of normal hearts will reveal to the interested examiner a great number of slight variations from what one had always considered "normal," and with increasing experience an examiner will learn facts and figures concerning physical signs for which he will search the text-books in vain. The absolute military necessity of training large numbers of medical officers in the late war for "cardiovascular" boards emphasizes the lack of appreciation for scientific accuracy in heart examinations in the general medical profession. If it was found necessary to especially train practitioners in this work for the emergencies of



war, is it not even more necessary that such men and similar practitioners train themselves to be accurate diagnosticians for the benefit of the public they serve?

We will now turn to the practical demonstration of methods of cardiac diagnosis. In order that time may be taken to emphasize details connected with physical examinations and with the physical signs found we will not detail the medical histories of the two patients to be examined, but will examine them as though they were unable to speak or understand our language.

Not the least important factor in an examination is the absolute necessity, if reliable diagnoses are to be made, of having the chest clothing removed, and not less necessary is it that the patient be placed in as good a light as possible. That diagnoses can be made without observing these precautions is true, but that such diagnoses can be as easily or as well made is unreasonable to believe. Whenever circumstances cause one to make an unsatisfactory attempt at a diagnosis with the patient partly clothed neither the physician nor the patient is satisfied with the result. Ideally, especially for women patients, one should have provided a loose-fitting kimona that can easily be lifted to expose portions of the body without interfering with the examination, while the presence of this garment will prevent the patient feeling too greatly exposed. One cannot, however, remind students too emphatically that modesty is a quality of the mind, and that no sensible patient objects to being thoroughly examined if the examiner is a professional gentleman and makes such an examination as free from unnecessary exposure as possible. We will now request that this young colored man remove his coat, vest, and shirt. The clinic is well heated and the exposure for the time that we will need him will not be harmful. He is now stripped to the waist, and you will agree with me that in attempting to make a cardiac diagnosis it would be folly to allow him to wear an undershirt unless the clinic were too cold for comfort.

Although we are particularly interested today in heart diagnosis, we will make a careful inspection of the head, face, mouth, teeth, and throat. The first visible abnormality that impresses

one is that the vessels of this patient's neck are pulsating violently in spite of the fact that the subject appears more interested in being demonstrated than alarmed or nervous.

Because of the natural pigment of the patient it is not easy to accurately state how cyanosed he is, but those near the railing can see that the patient's lips are somewhat dusky in appearance. The patient's facial expression is a little unusual in that his eyes are somewhat prominent, and the conjunctivæ seem suffused, as though he had been much exposed to the weather, which is not the case. When we have the patient open his mouth we see a fine set of teeth, not a tooth cavity being in evidence, and no teeth are missing. Those who are near can use this tongue depressor and observe two much enlarged and unhealthy appearing tonsils. One can but conjecture whether had these tonsils been removed a few years ago the patient might not have been in better health today. We glance at the chest and find a well-formed thorax. The patient has evidently lost weight, as he seems somewhat emaciated. We will have him sit in the middle of the clinic and continue the examination. To one making the examination it is clear that there is considerable bulging in the region of the precordium. When we glance at this area we notice at once a diffuse pulsation that is much more extensive than normal.

Each examiner probably stresses a particular part of the physical examination that he finds of greatest value, and to me the inspection and palpation of the heart and especially of the apex-beat gives more information than is obtained by percussion or auscultation.

It does not seem very important perhaps to inspect the cardiac area carefully for the apex-beat, to confirm its presence by the finger-tip, and then to really count the interspace very accurately, but when one sees an examiner carry out this very process one has much more faith in the conclusion arrived at than if the examiner ausculted the heart without this preliminary and, to a careful examiner, most necessary portion of the examination.

To remind students to use care in counting the ribs and interspaces seems perhaps uncalled for, but there are few students

who will, unless specially warned, count the ribs accurately. It is well to remember that the angle of Louis (junction of the manubrium and gladiolus) is always opposite the second rib and that the interspaces are always counted from the rib above. Another simple method of rib counting that many practitioners as well as students could adopt with advantage is that of remembering that the first rib below the clavicle that the examiner can grasp between the thumb and finger is the second rib. When one is sure which rib or interspace he is starting with the location of the apex-beat is an easy matter.

With our present patient the apex-beat is very diffuse, but its maximum point of intensity is in the sixth interspace, very near to the anterior axillary line. This dislocation of the apex usually infers one of two conditions. Either the heart is enlarged or it is displaced by the presence of fluid, air, or solid growth in the pleural cavity of the right side. We percuss over the right lower chest and find that this is resonant, so that we feel sure that the patient has no considerable amount of fluid within his chest. While palpating the apex-beat there was an opportunity of noticing several facts about the heart action. First, it was very strong and heaving in character; second, in addition to the marked pulsation at the apex there is a marked thrill.

Now a thrill is a vibratory sensation perceived by the hand when placed over the heart or over the blood-vessels, and is due to some alteration of the flow of blood through these organs. When we feel a thrill, the first question to ask ourselves is, Is this an exaggerated apex-beat, is it a functional thrill, or is it a thrill due to organic change in a heart valve that is obstructing the flow of blood in some direction?

Only by considerable experience in palpating cardiac areas in both health and disease will one be able to be sure in certain borderline cases. Fright, nervousness, overexercise, and certain changes from the effects of drugs can cause a "pseudo-thrill," physiologic or functional thrill, but, fortunately, the time of this thrill is always systolic in time so that it causes little practical difficulty in diagnosis.

It would be difficult to overemphasize the importance of systematic, thorough palpation of the cardiac areas for the presence (or absence) of thrills or shocks.

When a thrill is found as in the present patient's examination the question arises as to when in the cardiac cycle the thrill is felt. The determination of this matter is not always as easy as in the present case. I will have the patient walk to the railing, will have the first student place his finger upon the displaced apex-beat, and note each systole as represented by the lifting of his finger-tip. Now we will have him lay another finger flat in the interspace above the apex-beat where the thrill is best felt and see if he can feel the thrill. Now that the thrill is felt, the question arises, Does the thrill occur synchronously with the apex-beat or does it occur at a different time? Immediately it is seen that the thrill does not occur with the apex-beat, and therefore as there are but two portions to a cardiac cycle we know that the thrill is diastolic in time.

We now palpate over the body of the heart, and find that the thrill is as well felt over the body of the heart as near the apex. The heaving sensation is particularly marked over the fourth and fifth interspaces.

We now percuss out the heart borders. It is excellent practice in percussion, and without such practice no student or practitioner can learn to depend upon his percussion interpretation. Few believe that percussion is extremely accurate, and these few would be greatly disappointed if they confirmed their heart percussion by accurate methods that could not be questioned. Nevertheless, admitting the slight inaccuracy of percussion, the method gives us a guide to the condition of the right heart as well as to the left cardiac border, and the state of the aortic arch that makes the method well worth while. We find in the present patient that his right cardiac border apparently extends fully an inch to the right of the lateral border of the sternum, while the left border can be percussed well beyond the anterior axillary line in the fourth and fifth interspaces. These ink marks, although they do not show well, will indicate to those who are near the tremendous size of the heart that we are dealing with.

We will not percuss out the outline of the aortic arch, but will content ourselves with palpating the marked movement of the vessels at the root of the neck.

Thus far we have found by our examination a very large heart that causes a violent thrust against the chest wall, which we can see on inspection and better feel on palpation, there is a marked diastolic thrill over the body of the heart, and a somewhat modified thrill over the apex. As there is no question that the thrill is diastolic in time, we have a choice between a diagnosis of aortic regurgitation and of mitral stenosis, or of a combination of the two. In a heart so large we at once think of aortic regurgitation, and we feel the pulse to confirm the diagnosis. In this particular pulse one does not require to lift the arm above the body to find a well-marked collapsing pulse, which is an excellent example of the well-known Corrigan pulse. When we grasp the man's forearm one can feel the arterial pulsation all through the muscles of the arm. When the arm is lifted above the head the collapsing quality of the pulse is much exaggerated.

We press lightly upon the patient's finger-nail and see clearly the capillary pulse, which although found in certain other conditions of unstable circulation, is still most characteristic of the arterial changes in Corrigan's disease. If I have made myself clear you will note that to my mind the diagnosis is clear without our having listened to the heart, and one could prophesy in great part the auscultatory signs that can be elicited.

In the first place, one will probably hear a long-drawn-out diastolic murmur over the body of the heart, but especially about the fourth left interspace. We are likely to hear a loud systolic murmur at the apex which will be transmitted toward the left axilla. One could hardly imagine a heart whose left ventricle was so large in which the mitral leaflets would remain competent.

We have postponed, as you notice, listening to the heart until the last moment. This is, unfortunately, the reverse of the process usually adopted by many students and not a few physicians. To at once use a stethoscope in beginning to examine a heart is a most mistaken policy. It is far better to systematically

examine the organ in the way outlined above, and at the last moment to then auscultate the heart in order to confirm the opinion already formed from the results of inspection, palpation, and percussion.

As has been previously alluded to, it is essential that the examiner has had the opportunity of becoming acquainted with the normal sounds and the variation of these sounds as found in the varying forms of chests. Students are prone to believe that they can rely upon what they mentally decide is the first or second sound of the heart instead of confirming their opinion by either inspection or palpation of the apex-beat in conjunction with auscultation of the heart. If the sound heard is not synchronous with the apex-beat then it cannot be the first sound that is being heard, no matter how much the sound may resemble the first sound. Likewise, in murmurs as in thrills, although it may be at times easy to time the murmur or thrill without confirming one's opinion by reference to sight of the apex-beat or by palpating this best index of ventricular systole, still in the difficult case it is absolutely necessary, and until a student or a practitioner becomes very skilful (and after as well) it will be wise for him to use the precaution of placing his finger over the cardiac apex or over the carotid artery before he decides the time of any cardiac murmur. The heart that we are now examining is decidedly abnormal, for as we listen to the cardiac areas we hear a murmur at each of them. At the apex we hear the transmitted diastolic murmur from above, a little outside the fifth interspace we hear a loud systolic murmur from the mitral regurgitation that is the result of the enormous enlargement of the left ventricle, while the mitral valve leaflets must, of course, remain the same size, and therefore leakage back into the left auricle must take place. At the aortic cartilage and downward over the body of the heart is the loud diastolic murmur already referred to, and is due to the incompetence of the aortic valves. When the hypertrophied left ventricle has expelled a portion of the blood through the aortic orifice (a portion also leaking through the mitral valve into the left auricle) the aorta is distended, and at its recoil a portion of the blood surges backward toward the left ventricle.

As the aortic valves which should protect the heart from backward leakage are incompetent, the blood rushes through the orifice and causes the diastolic murmur heard.

When we listen over the pulmonary valves we at first hear nothing but the loud diastolic murmur, but if we accustom ourselves to this sound we will hear also the closing of the pulmonic valves.

At the tricuspid area (at the ensiform cartilage) we hear a loud systolic murmur which has a different quality than that heard over the apex. Another reason for being sure that this murmur is due to tricuspid leakage (leakage through the right auriculoventricular opening) is the fact that as we move the bell of our stethoscope away from the tricuspid area toward the mitral area the murmur becomes less distinct, while if we move the bell to the right the murmur can be heard for some distance.

Have we completed our examination? We have learned certain facts in connection with the patient's heart and we have done this while he has been sitting and standing. It should be a rule that when circumstances permit the patient should be examined standing, sitting, and when recumbent. It is a common finding that a cardiac murmur or thrill that is not in evidence when the patient is sitting or standing will become perfectly evident when the patient is placed in a recumbent position. This latter position too allows us to carefully palpate the patient's liver, and this examination should invariably be made in every cardiac disorder.

When we auscultate the patient's heart in the recumbent position we notice that the diastolic murmur is even louder than before, while the systolic murmurs do not seem quite as readily audible.

You notice at once that the patient is not as comfortable in the lying position and props himself upon his arms as well as the pillows to render his breathing easier. We can see even from a distance the enlargement of his liver, and if I place my hand over the right lobe and ask that he stop breathing a moment you can all see the pulsation of my finger as the liver strikes against it.



We have examined this man hurriedly so that you can visualize the method. There are many other things that we can do to make our examination complete. We have already had certain of these helpful diagnostic aids performed in the dispensary. His systolic blood-pressure is irregular. At one estimation it was noted to be 200, while a number of estimations have shown varying pressures of from 160 to 210. This means that the patient's myocardium is, of course, the cause of his present discomfort, and this is true in the greater number of so-called valvular heart cases. We wish you to know the signs connected with valvular deficiency, but we wish you to ever keep clear in your mind that the greater amount of attention must be devoted to the myocardium in diagnosis, prognosis, and treatment.

I am anxious for you to remember that if you will be content to examine a patient in the way that we have done before you today you will be in a very much better position to understand what is taking place in the heart and at its orifices than if you make your first act to listen to the murmurs that are at best a poor index of the heart's disability.

One last sign that it will be well to show you, and that is that he has only a slight amount of edema of the extremities, and that he has no gross amount of fluid in his peritoneal or pleural cavities.

The patient's urine will be examined when he reaches the ward and a Wassermann test will be made upon his blood, as should be made upon all the bloods of patients with chronic diseases. There is no reason for thinking that the cardiac condition is other than a rheumatic endocarditis, but all the necessary tests must be made. We do not wish to guess or to teach you to guess, but rather to prove and to teach you to prove your work in every practical way. We will have the patient sent to the ward where he can be made comfortable by several therapeutic measures. A thorough blood-letting would be an excellent bit of therapy and will bring about a much needed rest and sleep. He will require no opiate if he is bled, but if this is not done he will be the better for a hypodermic of morphin as soon as we know exactly what state his kidneys will be revealed



to be in by the urine examination. Digitalis will be helpful, but as the patient has been taking large amounts of this drug for some time past it will do him less good, in my opinion, than the bleeding and opiate. Later digitalis will be the sheet-anchor of treatment.

We will now have the second patient admitted. This young woman is making her first visit to the hospital, and some of you may be inclined to regard the pink flush on either cheek as an evidence of excitement or possibly of the use of cosmetics. It is neither in this instance, as the patient assures us that she has been flushed as you see her for quite a period. You will note her red lips which are almost crimson. Again, this is a characteristic that the patient has noted for some time past.

Now comes the question as to whether the patient, being a timid gentlewoman, should cause us to alter our method of examination because she is a woman. Most sensible women, as in the excellent example before us, state that whatever is right and necessary they are willing to have done in the way of examinations, and so I have had the clothing of the chest removed and the upper body covered with a light blanket so that at the same time the patient's feelings are spared and still a thorough examination can be made.

We have inspected the lips and cheeks, and on further inspection you will note a rather drawn look in the face and an anxious expression about the eyes. The scleræ seem unusually clear and somewhat moist. When we glance at the neck we again see pulsation of vessels, but of quite a different type than was noted in the last patient. In the last patient the vessels could be grasped and it was easily seen that the vessels were throbbing arteries. In the present patient it is the veins and not the arteries that are so much in evidence and that can be seen to pulsate.

The chest is slightly formed and the patient states that she has recently lost 10 pounds in weight. We notice quite a contrast in glancing at the precordium from the condition that that we observed in the last patient. Here we find an ill-defined apex-beat within the normal line, being in the fifth interspace,

and thus showing no enlargement of moment. As we place our hand over the apex we notice two characteristic signs. The first is a sharp, systolic shock or tap. This comes just as the apex comes against one's finger and thus can be easily timed. The second sign is a marked thrill that comes immediately before the apex-beat. This is often spoken of as a presystolic thrill, but we must remember that presystolic thrills and murmurs must occur in diastole and are therefore diastolic thrills and murmurs, although to many the word "presystolic" gives an added significance in that the examiner knows that such a thrill or murmur occurs during *auricular systole*. As you will remember from your studies in physiology, the blood enters the ventricles during diastole largely by suction of the relaxed ventricle drawing the blood from the auricle. When, however, the greater portion of the blood has been admitted by suction, the last remaining portion is thrust out of the auricle by auricular contraction. It is at this period of the cardiac cycle that the so-called presystolic murmurs and thrills are heard and felt. This thrill is felt distinctly before the apex-beat and stops abruptly with this sharp tap of the apex-beat. What could cause a diastolic thrill at this area? Only two common conditions. One is aortic regurgitation, that we have seen in the last patient. We can rule this condition out by noting first the size of the heart, second, the character of the pulse, which in the present patient is small, easily compressible, which is a great contrast from the character of the pulse of the last patient. Again we note that the thrill is limited to the mitral area and is not felt over the body of the heart, and lastly, when we listen to the aortic area, there is heard a normal sound over the aortic valves revealing the fact that these valves are properly closing.

The second commonly met with heart condition that can cause a diastolic thrill at the apex of the heart is mitral stenosis. This is, to my mind, the most important cardiac lesion to discover and to thoroughly understand. I believe that this particular lesion causes more difficulty with most students than any other, but I think the reason is again because the student uses

the stethoscope too much and his eyes and his hands too little in making the examination.

We now percuss out the heart borders, and we find that the right is slightly enlarged beyond the lateral sternal border. The left ventricle is slightly enlarged by percussion, although the apex-beat is practically in the normal line. On auscultation we hear a rough, churning-like sound that ends abruptly in the first sound of the heart. The murmur is, of course, caused by the same condition that causes the thrill, but sometimes in obstructive lesions the thrill is more easily felt than the murmur is heard, and, on the contrary, in many regurgitant murmurs, especially at the mitral and tricuspid areas, the murmur that is perfectly easily heard causes no palpable thrill. The reason or reasons that govern the laws for the transmission of vibratory sensations within the chest have not been accurately determined, but has no doubt to do with the media through which the vibrations must pass from their source of origin to the palpating finger or hand.

The murmur of mitral stenosis is less likely to be transmitted to a distance, but is usually found to be somewhat transmitted, as in this case, upward and to the right. When we listen to the pulmonic sound of the heart we find that this is greatly accentuated when compared with the aortic sound. It is to be remembered that all cardiac pulmonic sounds in youth should be slightly louder than the aortic sound at the same age, but in this instance the valve leaflets of the pulmonic orifices are being violently vibrated and closed together by the pressure within the vessels of the lung. We are evidently here dealing with a case of fairly well compensated mitral stenosis, and when we stop to think of what must become of the blood that is unable to enter the left ventricle we know that it must pass from the left auricle back into the pulmonary system which is already overfilled with blood from the right ventricle, we can easily understand why there is an accentuation of the pulmonic sound of the heart.

There is one excellent reason why so many men who listen to such a heart as this patient has are deceived as to the true nature of the cardiac lesion. After this patient exercises if one listens

to certain portions of the chest about her mitral area they will hear a clear and undoubted mitral regurgitant murmur, and, of course, this is systolic in time. If one jumps to the conclusion that the systolic murmur is all that is at fault in the heart they will be much deceived. If the examiner has been careful in applying his knowledge of inspection and palpation he will be fairly sure that the patient has a mitral obstructive lesion long before he listens to the heart, and if he hears only a systolic murmur he will move the bell of his stethoscope about the mitral area until the diastolic murmur is found. If the murmur is not in evidence he can accentuate it materially by having the patient take a number of deep inhalations and then hold the breath for a few seconds. This method is worthy of much more prevalent use, as is the advantage of various postures to accentuate both thrills, murmurs, and shocks. A slight amount of exercise in the usual case will accentuate the physical signs to a marked degree, and if the patient is physically fit there is no harm in such exercises; in fact, in obscure cases they are of the greatest possible help. A patient who is in bed can bend over quickly a few times and increase the circulatory rate to such a degree that a practically latent murmur will appear very distinctly.

We will have the patient now placed in a recumbent position, and the first observation to be noted is how much more distended the veins of the neck are while the patient is in the recumbent posture. If the vessels were arteries the pulsation would be more evident when the patient was standing than in the recumbent posture. This patient, too, is not at her happiest in attempting to lie with her head low. This patient and the former one is of the class that occupy bed-rests in the wards. We find that in the recumbent posture the thrill is a little less evident, while the murmur to be heard in the area where before we heard the churning diastolic murmur is now systolic in time. If, however, we listen higher up we find the crescendo murmur rough and rasping and ending in a loud first sound; as is proved by our finger upon the apex or upon the carotid. While the patient is recumbent we palpate the liver area and find that the patient shows an expression of pain when we place our fingers too deeply

under the costal margin. When she takes a long breath the finger comes in contact with the liver and the patient again winces from discomfort. If we ask the patient to stop breathing again for a moment we again note a pulsating liver. This is not as evident as was the last patient's pulsation, but is quite marked enough to impress us with the fact that there is too much blood in this patient's liver.

There is, as you can see, edema of the lower extremities of slight degree, and the same treatment as was advised in the last patient will prove helpful. I have shown you, gentlemen, an example of the two most important cardiac lesions. No one is better aware than myself of the superficial character of the demonstration. There are many interesting features that could be discussed in connection with both patients, but the point that I am most anxious to impress upon your minds is that in order to make a correct diagnosis it is absolutely necessary that a systematic examination should be made, and that the most important part of the examination comes long before the use of the stethoscope. If a student or a practitioner does not feel pretty confident of the diagnosis of a cardiac condition before he listens to the murmurs that are to be heard he is not likely to be correct in his interpretation of the murmurs he does hear. The experiences of the war has taught us much in cardiac diagnosis, and it has taught us nothing that compares in value with the recognition of the variation from the supposed normal that had been commonly regarded as more or less fixed. Early in the war it was learned that if the examining officers were to rule men out of military service because they had systolic murmurs either at the base or the apex of the heart that it would take a much longer time to gather together an efficient army than was thought. It was only a few months after our belated entrance into the war that the Surgeon General's Office issued new orders for dealing with the cardiovascular examinations. It was then ruled that if one was dealing with a normal sized heart that all systolic murmurs could be with safety ignored, and this proved to be a very wise policy.

We can use this method of examination in private life with

great advantage, for it is the murmurs that occur in systole that attract so much attention at the base of the heart, and at the apex that are due to functional and unimportant conditions. On the other hand, all diastolic murmurs are worthy of careful study and usually require a modification of the patient's activities.

Learn to use your eye, your finger, and your hand in examining hearts, and use the stethoscope only to confirm what you have already learned by the more reliable and useful examinations.

## ETHICS, IDEALS, AND EFFICIENCY IN THE PRACTICE OF MEDICINE <sup>1</sup>

GENTLEMEN:

Owing to the unavoidable absence of Professor Hare due to a disabling but (fortunately both for him and for us) not dangerous illness he has requested that this hour and, if his continued incapacity necessitates it, next week's similar hour be occupied by me.

As this is the first clinic of the present session it will not be out of place for one who has welcomed the arrival of a number of classes of senior students to the front benches of the amphitheater and welcomed the members of the junior classes to the more modestly retired places to bid the men present a most hearty welcome and to wish for them a very pleasant and successful winter's work. It will be good to see you about the wards again for the hospital needs, as do your teachers, the stimulating inspiration of your presence in the class-rooms and in the clinic.

During this introductory hour instead of bringing patients before you I wish to briefly consider with you certain ethical principles that should influence our ideals as medical students and to more fully emphasize the great necessity of efficient methods in both the study and practice of medicine. If as undergraduate students you form correct and lasting impressions concerning the obligations and responsibilities of each member of the medical profession, it will not only be well for you but for the profession of which guild you will soon be active members. It is well to touch upon such an important matter as medical ethics at the beginning of the college year lest in our routine study of the practical details of clinical medicine we fail to keep distinct in your minds the highest ideals of the profession.

<sup>1</sup> A Clinical Lecture delivered at the First Clinic of the present session of the College, October 6, 1919.

One sometimes hears the term "ethics of the medical profession" ridiculed and smiled at as an example of impractical idealism, but never by thoughtful, understanding men or women.

Ethics, as you know, is the science of human duty, and thus in a consideration of medical ethics we learn not only what specific duties we are under obligation to perform, but how best to perform them in conjunction with other physicians, with the relatives and friends of the patient and with the general public, so that our behavior will always reflect credit upon ourselves and upon our profession.

In Section 1 of the Principles of Medical Ethics adopted by the American Medical Association, in which every worthy medical student should look forward to deserving a fellowship, we read, "The practice of medicine is a profession. A profession has for its prime object the service it can render humanity; reward or financial gain should be a subordinate consideration. In choosing this profession an individual assumes an obligation to conduct himself in accord with its ideals."

You will note that financial gain should be a subordinate consideration. This means that when we become more interested in the contents of our patient's pocketbook than we are in studying his physical condition we need to reread and inwardly digest Section 1 of the Principles of Ethics of our own profession.

What, then, are the ideals of our chosen profession which each one of us is obligated to conduct himself in accord with?

The most important ideal for a physician is that which was given to the world nearly two thousand years ago and is best epitomized in the Golden Rule: To do to others as we would that they should do to us. If we live by this rule we will be a credit to our parents, an honor to our profession, and unusually helpful citizens.

The simple virtues of daily living are the ones most necessary for each of us to cultivate. To be honest; to be unselfish; never to be too busy to be kind; to be charitable; to be generous to those who need our generosity, and to cultivate "that only ambition that never can become a vice—to excel others in doing good."



These are the traits that we can all attempt to cultivate for our souls' good and for the honor and fair reputation of our beloved profession. It is well for each student and each physician to think of himself not as an individual in the profession, but as a fractional part of the profession in any town, county, state, or nation.

If a student or a physician does his work as well as he can do it he increases very materially what a baseball fan might term the "batting average" of the medical profession for his particular section of the country. On the other hand, if one is content to perform careless, indifferent work, how easily he becomes classed with those who have a low average for medical efficiency.

Were every practising physician throughout this country well trained, interested in his profession, and efficient in his work there would be little opportunity for the men before me to establish themselves. You need, however, have no fear for your future in the profession. The country has need for every well-trained, conscientious physician, and success lies before each man if he will but deserve it.

It is well to remember that the best kind of success in medicine can only be built upon a firm foundation of systematic preparation and a never-failing interest in well-conducted, practical scientific work. Too rapid success may prove a very real danger in that it may tempt a physician to exchange the ideal of best quality of service for that of the greatest quantity of service rendered.

You have already been impressed, I feel sure, with the truth of the statement that in the profession of medicine one must, of necessity, if one loves his vocation, remain a student.

There never comes a time in a physician's life when he does not require, and if he is wise, does not use, a student's lamp. Neither does there come a time when reading and study do not reveal new light being thrown upon the problems of medical science by the investigations and observations of the inquiring minds of the students of yesterday and today. It has been truly said that a science that does not progress rapidly becomes a dead science. It is certainly true that in the science and in the

art of medicine there must be constant alteration and improvement in the theories, methods, and technic to keep pace with the discoveries and advances of the closely allied sciences.

These facts indicate that we must be wisely industrious both as graduate and undergraduate students, remembering that in very truth the graduate and the undergraduate are students together, even if a few or many years separate their ages and medical experience.

The adage "Experience is the best teacher" is only true in medical work if we make sure that we learn all there is to learn from the daily lesson of "experience." The hearts and brains of all men or women trained or being trained in medicine are inspired by the same divine fire that causes an unquenchable thirst for such knowledge as will be of service to diseased humanity.

Were one to ask each man present why he had chosen the field of medicine for his life-work the average answer would be, I think, that he wished to enter an honorable profession, wished to be useful to his fellow-men, and wished to earn a competence for himself and for those dependent upon him. What are the characteristics of the medical profession which have caused it to be universally recognized as an honorable profession?

Has it been the ability of its members to accumulate large sums of money? It is a well-known fact that for the time and study required and the money expended in preparation for the practice of medicine and for the amount of energy, industry, and self-sacrifice necessary to live the life of a physician the financial return compares unfavorably with other professional incomes, and is ridiculously small when compared with the earning capacity of a modernly trained business man.

The course of study necessary to give a medical student the proper foundation for the actual practical study of medical science that fills his professional life from the time of his graduation is by far the most severe scientific course that is known. You men, perhaps fortunately, have little time, desire, or opportunity to compare the courses of study of the various professions, but you have, no doubt, noticed that the course in medi-

cine is not popular with those ease-loving students who are intent upon combining recreation and amusement with their professional studies. Those of my hearers who are sons of physicians or those who are intimately acquainted with the daily routine of a physician's life need no word of mine to remind them that the members of our profession do not escape long hours of labor, and that even their hours of relaxation are not free from the anxieties that are so closely connected with our work. Young as you are in the profession you are well aware that there are neither union hours nor a fixed wage scale for physicians, and one seldom hears of physicians striking for less hours or for larger fees. No true physician ever refused to aid those who required his services because they lacked the means to pay. Every physician expects and desires to treat those who are unable to pay for treatment or directs them to agencies where they can be treated.

Are you not proud that we belong to a profession that supplied over 30,000 physicians for the urgent needs of the military and naval services of our country during the recent war, and also supplied an equal or even larger number for the important professional duties connected with the draft boards and the Red Cross agencies as well as other similar activities.

You will agree with me then that the reason our profession is termed "honorable" is that, at heart, all true physicians are idealists, and the members of our profession most loved and admired by fellow physicians as well as by their patients and the general public are those most unselfish, most self-sacrificing, and most anxious to serve to the best of their ability those who seek their aid.

Such physicians are an honor to our profession, and their reputation and example does much to neutralize the poor opinion that the public sometimes forms of our profession through their misfortune in coming in contact with certain physicians who look upon their profession as a "business," and whose only interest in it is the financial return that they can obtain.

We have spoken of the ethics of our calling and of the ideals that should stir our hearts with the desire to be honest, helpful,

and true physicians. Now let us consider for the remainder of the hour how we can best cultivate methods of efficiency in our profession.

We have all heard, especially during recent years, the terms "scientific management" and "efficiency" as used in the world of business. We are aware that there is scarcely a modern commercial enterprise or corporation that does not employ its efficiency expert or experts. The sole duty of these specialists is to make a scientific study of the methods for increasing the efficiency of each human or mechanical unit that goes into the make-up of a twentieth century business.

If it has been found necessary in the commercial field to have an efficiency expert point out vulnerable spots in business systems, how much more necessary it is that we medical students and physicians, whose daily work will be potentially far more valuable to our patients than mere money making, should scrutinize most carefully our methods of study and work and test the efficiency of both our work and our study in every possible way. Those of us who read the popular magazines, especially those devoted to business interests, cannot but have formed some general idea of what is accomplished in any line of work when a practical expert teaches a workman to correct faulty methods of work, eliminating waste motions, and accomplishing his purpose with greater accuracy and with less fatigue. It has been proved many times, and to the entire satisfaction of even unfriendly critics of the so-called scientific management, that the adaptation of the practical results of such careful studies and methods can and do increase greatly the efficiency of even skilled workmen. As a profession we are not fond, perhaps, of thinking of ourselves as business men, but it is not improper that we should consider ourselves skilled laborers in our own field and study how we may increase the efficiency of our studies and daily work.

How many students or physicians would enjoy having an unprejudiced medical efficiency expert accompany them in their daily routine of study in the case of the undergraduate or the daily professional work in the case of the graduate physician for the purpose of reporting upon their efficiency as medical students on

the one hand or as public servants on the other? This would mean that such an observer would be at the elbow of the student in ward, class, and clinic, in lecture hall, and during the study hour; while similarly such an observer would be with the physician during office consultations, watching both the physical examinations and the laboratory work, accompanying him on his round of visits and continuing during the periods that could be devoted to reviewing the literature concerning some obscure condition encountered during the day or consulting standard text-books for further information.

How few of us, either undergraduate or graduate students, would look forward with pleasure to reading such an expert's friendly but just criticism of our methods of accomplishing our daily duties. The thought of each undergraduate having such a report signed, let us say by Professor Hare or Professor McCrae, and each graduate receiving a similar report signed by the Carnegie Commission brings clearly to our minds the unpleasant realization that many of us would not measure up to a high standard of efficiency if we were judged by strict standards. The low standard would be due, in the majority of instances, not to ignorance or even to faulty training, but to carelessness and lack of a proper and systematic routine.

The undergraduate is prone to imagine that he will change his methods for improved ones when he graduates, but the truth is that few men change their methods of thought, study, or work after graduation. It is a disagreeable thought, but can we deny that frequently our methods of work and study as senior students are not those that we would highly recommend to the less advanced students, and as graduates and practitioners we do not always adopt and adhere to methods of painstaking diagnosis that we would wish employed by fellow practitioners if we or members of our families were the patients being examined. That it is wise or at least advisable to recognize our own deficiencies we all know, but it is a trait of human nature to avoid, if possible, a consideration of one's weaknesses until they are called to our attention.

An excellent illustration of the good accomplished by just

criticism was the effect of the report issued in 1910 concerning medical education in the United States and Canada by the Carnegie Foundation for the advancement of teaching. Although there were other important influences in bringing about the important and necessary changes in the methods of teaching in the medical colleges of this country, there was no one influence so potent as was this unbiased critical report. Since that report was published there has occurred important advances in medical teaching. It is much easier to endure criticism from a fellow-student than from an outsider because the critic can hardly be thought to be free from the faults that he discloses. Let each of us ask himself the question, Do I do my work as well as I can? Do not some of us as students drift more or less aimlessly through our courses, forgetting that if we do not adopt and use an efficient system of work and study during our undergraduate days that we are never likely to pursue any other in our later professional life. As graduate physicians will not such men practice at times a system, long since discarded in the legitimate business world, of having more than one price for their services, and would not the ancient maxim of the auctioneer, "let the buyer beware," be applicable to such services when professional work is carelessly done.

When as resident physicians men do more careful work and with better spirit for the patients in the private rooms than for the patients in the wards the professional feet of these men are straying into dangerous paths. If in an emergency it seems necessary to neglect any patient, let us see to it that the defenseless ward patient or the patient without means be not the one to suffer. The patient with education and financial resources can obtain other professional care without difficulty, while those possessing neither resource are at the mercy of their medical attendant. The grossly unfair part of the system where a physician gives better service for a large fee than for a small one, or gives better attention to the man or woman who has the education or intelligence to demand it, than to the ignorant, is that many patients are much handicapped by their ignorance as to what they have a right to expect for the fee they pay a physician.

The prestige of being a member of an honorable profession, a large proportion of whose members attempt to give full value for the money received for professional services, carries many a practitioner along when he may be doing very careless and indifferent work. A medical diploma and a license to practice medicine was never intended to be used to deprive citizens of their money without giving full value in skilled scientific services in return. The term "obtaining money under false pretenses" could apply to many practitioners of medicine that do not attempt to study their patients as is absolutely necessary if the patient is to be relieved. A pleasing personality is certainly a desirable quality in a physician and the fortunate possessor is envied by all, and still the possession of such a winning personality sometimes induces its potentially lucky possessor to neglect the every-day duties of his profession (physical examinations, laboratory tests, and careful study of his patients) for the reason that he is able to hold his patients with pleasant speeches and with lamentably little effort in the way of scientific investigation and study. When a patient visits the office of a physician or is visited by a physician the patient is fully aware that the state has authorized the physician to practice the art of healing, but such an individual must often be puzzled by the lack of uniformity in the quality of services rendered by different members of the profession. Intimately connected with the question of how much service shall a patient receive for his fee is the inquiry, Shall the amount of the fee determine the quality of the service?

For every true physician there can be but one answer. If he assumes the responsibility of treating a patient, then, regardless of fee, he must give the best services possible. A doctor can decline to assume charge of a patient, he can refer the patient to others, but if he takes the patient under his care he should give him his most skilled services.

Anyone who has been in a position to observe the tendencies of the relations of physicians and their patients and particularly to observe the mental attitude of the public toward physicians in general, cannot but have felt that during recent years there has been a tendency for the physician to find his place of honor



in the affection of the public less secure and even to find himself displaced by osteopaths, mechanoneuropaths, chiropractors, Christian Scientists, and other unscientific impersonators. That there are natural causes for the change in attitude of a certain proportion of the public toward the physician will not be questioned or discussed, but the most important cause lies in the professional shortcomings of physicians. The attitude of the general public toward the profession of medicine is usually very loyal until it awakes to the fact that it has been deceived or cheated. Can any of us doubt that the loyalty of patients will be sorely tried if we do not invariably give them our best professional work? If every man before me gives to his patients his best services at all times there will be little demand for the services of charlatans or deluded sectarians in medicine among his patients. It has been the careless methods as well as the neglect of a proportion of licensed practitioners of medicine to study scientifically the chronic illnesses that has driven certain patients to consult those whose lack of knowledge or lack of honesty causes them to promise the patients much that an honest and thoroughly trained physician would not feel justified in stating.

The physical methods in common use by such irregular pseudo-scientific practitioners could be used with far greater benefit by physicians who would take the time and make the necessary study and investigations to familiarize themselves with the best methods of dealing with certain common and chronic disorders.

Because a physician possesses a license issued by the authorities of the state that entitles him to practice the art of healing is all the greater reason why he should give full money value for the fee received from his patient. We owe this not only to the individual who consults us, but we owe it equally to the Commonwealth which issues our license. In such a hospital as the Jefferson we come in contact with large numbers of patients who are suffering from chronic illnesses. To listen to their medical histories either makes one very glad that he belongs to the same profession as did their attending physician or makes one regret that the individual who attended the patient was a member of our profession. Many patients who are obviously suffering

from far-advanced chronic disorders and who have been under the care of physicians for varying periods of time, state that they have neither been examined nor have the necessary laboratory examinations been made. Many of these patients could have been saved months if not years of invalidism and, in certain of them, their lives might easily have been saved had a careful physical examination and the necessary laboratory tests revealed the nature of the difficulty and suitable treatment been adopted.

Often it is possible to talk with the physician in attendance and to learn from him why an examination was not made. The most common excuse is the one that I trust you will never use to explain a neglect of professional duty. The excuse is that the physician did not feel able to examine the patient for as small a fee as competition compelled him to adopt. Another and similar excuse is based upon the same principle, namely, that there is not sufficient time in the day to examine all the patients who consult a physician. Are such excuses valid when carefully studied, and is the patient, under such conditions, informed that his case has not been thoroughly studied?

We must turn to Section 2 of Article 6 of the Principles of Medical Ethics to obtain the official answer, and there we find that "it is unprofessional for a physician to dispose of his services under conditions that make it impossible for him to render adequate service or which interferes with reasonable competition among physicians of the community. To do this is detrimental to the individual and lowers the dignity of the physician." It might also be stated that such a plan is most unjust to its immediate victims, the patients, who deceive themselves by thinking that their lives and health are safeguarded by the physician when, as a matter of fact, he may neither know the cause of their symptoms nor does he make any scientific attempt to learn the nature of the illness. One must bear in mind that the physician who sees a patient for a small fee often sees him frequently, and if such a patient is seen a number of times during a short period of time he is paying a high price for inefficient services unless he receives a thorough examination, has his case properly diagnosed, and has proper treatment instituted.

The physician who fails to do good, careful medical work is doing himself, his patient, his profession, and the state a grave injustice.

It is infrequent for any one physician to see a large number of *new* patients in a single day. It is the patients whom we have seen previously who occupy the greater number of places in our offices, and it is well to remember that if the patient has been carefully examined and the proper records made and preserved, the percentage of patients requiring complete re-examinations is comparatively small.

What, then, is the duty of a physician, whether he be serving as hospital physician or as a private practitioner, when he sees a patient for the first time? If he is to live up to the obligation of being a member of an honorable profession he cannot do less than he would wish done if he was the patient and the patient was the physician.

You have learned long since that a correct diagnosis depends upon many factors, but perhaps none is more important than a complete history of the patient's previous history, medical and otherwise.

Such a history should be taken, brief, but complete in the essentials. This record should be preserved in such a way as to be easily accessible, for it is of very real value, and will save far more time and possibly be the means of bringing to the doctor more money than he could possibly lose by taking the time to make the record.

A thorough physical examination of the patient with the clothing of the chest removed should be made to reveal the state of the most vital organs. A record of the result of this examination should be included both as to negative and positive findings. This record should include such essentials as the weight of the patient, the pulse-rate, the temperature, and, if possible, the result of the urine examination and the blood-pressure estimation. In all chronic disorders of patients whose histories you are not familiar with it is an excellent rule to add to the above record the result of the Wassermann test, and, when necessary, a complete blood examination.

It is more than possible that some of the men before me will consider certain of the examinations that I have advocated unnecessary and possibly impractical in every-day office work. Some will think that such a plan of procedure can only be adopted by physicians who charge large fees. This attitude of mind is a mistake, for every physician is compelled, whether he wishes to or not, to devote a certain amount of time to his patient in order that the proper psychic effect will be obtained. Too often physicians, especially those who are physically and mentally indolent, spend this necessary time in talking to the patient and thus impressing him that his case is understood, when, in reality, the patient is receiving little that is scientifically helpful in his consultation.

Nothing but good can result from the physician spending the time that is devoted to the patient and for which the patient pays in carrying out scientific investigations, such as studying the pulse, recording the temperature, estimating the blood-pressure, and, if necessary, examining the patient completely.

It takes relatively little time to accomplish these acts, and when they are completed not only is the patient better pleased, but the physician is definitely aided in his search for the cause of the symptoms. One can well occupy the time when the patient is dressing in examining the urine and with the exception of the microscopic examination this can easily be completed by the time the patient is dressed. There are those who consider the time wasted in examining organs in which no pathologic lesion will be found. It is true that the patient is not directly benefited by such an examination, but it is also true that it is a great satisfaction to the patient if he can be assured that his vital organs, *when carefully examined*, reveal no evidence of disease. To the physician and especially to the young physician great advantage is derived from the information obtained by such painstaking examination, as this is the only practical method to become intimately familiar with the normal conditions of the body, and thus through this knowledge to be able to recognize slight alterations.

The examiner who examines only those patients who seem

to him to require an examination is doing both himself and his patient an injustice. Unless he is a most unusual man he will often fail to recognize the early symptoms or physical signs of a disorder, and will often not be able to discover the earlier and more easily corrected stages of a disorder because he is not in the habit of making routine examinations, and is likely to only perceive those marked changes that do not yield readily to treatment.

In deciding the quality of work that we intend to offer to the public we should ask ourselves the questions that follow: Is the physician who examines completely one or two patients a week likely to detect an early pulmonary lesion? Is he likely to correctly interpret the rapid pulse associated with tenderness and rigidity in the right iliac fossa in the patient who comes to his office with a long story of chronic indigestion with a recent attack of acute abdominal pain? Does the presence of a thrill at the apex of the patient's heart cause him to examine that organ most carefully and, if necessary, to warn him of the possible dangers of overexercise and thus guard the health of a cardiac patient?

In all these instances the life and the future health conditions of the patient depend very much upon the professional advice of the physician in charge. Is it stating the matter too strongly to say that every physician controls the destiny of certain of his patients?

It would be difficult to emphasize too strongly the necessity for routine urine examinations in every-day work. Whether one has a Bunsen burner or an alcohol lamp it is an easy matter to add a small quantity of dilute acetic acid to a test-tube of urine and then to heat the upper third of the tube and observe whether there is a clouding in the heated portion compared with the unheated. The time required to make the examination is trifling, but this simple examination has potentially saved more lives from invalidism and even death than one is likely to think.

One or two minutes suffices for a routine examination of the urine for sugar, and then the urine may be set aside in a sedi-

menting glass to await the convenience of the examiner to search the sediment for evidences of pathology. The time necessary to make this last examination is slight, for the gross pathologic changes, such as pus, renal casts, and blood-cells, are quickly recognized. In the patients whose histories or whose physical signs are suggestive of renal changes it may be necessary to make a longer search. If one is tempted to consider that urine examinations as a routine are unnecessary, it is well to ask the question Where do all the chronic, unrecognized cases of nephritis come from?

Such patients when questioned at the dispensary often state that they have been treated for long periods for "anemia," "indigestion," "headache," and "dizziness" without their urine having been examined and without any advice that would lead one to believe that the nephritic condition had been suspected.

Why do you suppose so many of our patients who reveal upon their first examination a severe crippling of the heart valves and deficiency in the muscle have been treated for "liver trouble," "indigestion," and "stomach trouble," without having been warned of the dangers of overexercise and without having been examined?

Under whose care have the patients been who reveal far-advanced tuberculosis, but who state that they have been treated for "stomach trouble," "chronic bronchitis," "chronic pleurisy," "heavy cold," etc.? Who but the physician or physicians that they have consulted in the past are accountable in certain instances for the patient's feeling of security in that their illness had been pronounced "nothing serious" without a physical examination having been made, without an estimation of the temperature, without the pulse having been counted, or even a careful history having been taken when an examination reveals far-advanced pulmonary tuberculosis. It is scarcely necessary to remind you of the cases of irregular bleeding in women, especially at the time of the menopause, who have been treated "medically" without a pelvic examination, and who, too late, are discovered to have malignant disease of so advanced a character as to preclude surgical treatment.

We see far too many cases of visceral lues that are allowed to progress and sometimes allowed to become very ill without having had a physical examination and without the recognition of an often curable condition.

In many of the states with progressive Health Departments a physician can now have a Wassermann test made upon the blood of any patient without cost to either the patient or the physician, and in all obscure conditions we should all make use of this opportunity for disproving the presence of this, always possible, infection.

All the above statements are to direct your attention to the fact that in medicine the practitioner is most successful who has the best routine system and who adheres to it faithfully, making such changes as additional experience points out as wise and necessary.

To train our eyes, our hands, and our ears is very essential, for as Keen well says: "With all our varied instruments of precision, useful as they are, nothing can replace the watchful eye, the alert ear, the tactful finger, and the logical mind which correlates the facts obtained through all these avenues of information and so reaches an exact diagnosis." The system of medical education is constantly improving, and each class of students that leaves the school goes out a little better prepared than the classes that preceded it. What does the new graduate require to make available his store of knowledge? He needs daily experience with patients and intimate contact with human nature. Each patient that he makes a thorough study of will make the next patient's examination and treatment not only easier, but much more valuable. Skill can only be acquired by doing the same act frequently and by learning something new each day. To test the pupillary reaction, to observe the arcus senilis, to glance at the pinnae of the ears for the presence of gouty tophi, to examine the teeth, the tonsils, and the thyroid gland as well as the ear drum of every patient seen for the first time may seem a waste of time to those who are restless to see the next patient, but all these tests can be done very quickly and have proved most helpful to those who make such examinations a routine.



Without such routine examinations it is quite possible that a patient will suffer for weeks or months without the true nature of the disorder being revealed.

If every man in this clinic will make it a fixed rule to examine the throat of every child whom he sees professionally, no matter what the symptoms complained of may be, he will never overlook an unsuspected diphtheria, a follicular tonsillitis, or a Vincent's angina. If each man makes a similar rule that he will examine the skin of every child whom he sees professionally he will never have the humiliation of having some fellow practitioner recognize one of the exanthemata that he should have recognized. Another equally important rule to be strictly adhered to is that concerning the inspection of the ear drums of any child who is ill, particularly in those sick of an unexplained fever, or in adults who are unconscious or partly so. This examination will save many children unnecessary suffering and prevent just criticism of our profession. If we examine the urine of all young children microscopically and especially in those cases of undetermined types of fever, if we will examine the urine by cultural methods we will learn the true nature of more than one puzzling illness.

This clinic, fellow students, may have sounded too much like a sermon, but the excuse for so much advice lies in our being members of a profession that imposes unusual responsibilities and obligations upon each of us. We stand between our patients and death or, what is far worse in many instances, between the patient and chronic invalidism. I am deeply interested in the success of every student of the Jefferson Medical College, and I am equally interested that the college as well as the medical profession be represented by men whose professional and ethical standards are of the highest.

Let us each one see to it that it is through no neglect or carelessness of ours that a patient drifts into an unrecognized pathologic whirlpool.



## CLINIC OF DR. H. K. MOHLER

JEFFERSON HOSPITAL

### DISCUSSION OF DIABETES MELLITUS IN CHILDREN

DIABETES mellitus was first recognized and described by Thomas Willis, an Englishman, in 1674, as a disease with sugar in the urine as its chief symptom.

It was not until a century later, after Dobson and Pole had succeeded in demonstrating directly the presence of sugar in the urine, and John Rollo had confirmed the injurious effects of starches and sugar in the course of diabetes, that physicians began to take a keen interest in this disease.

The symptomatology of diabetes mellitus has been well understood and recognized for many years because of the intensive study that this disease has received.

The etiology has always been the subject of much speculation, and even today opinion is not united as to the etiologic basis of the disorder of carbohydrate metabolism. With the rapid strides made in physiologic chemistry much has been brought to light with regard to the changes that follow the ingestion of carbohydrate in the normal individual and the diabetic individual. Upon this information has been developed, most notably by Allen, a rational treatment which is productive of the best results in patients suffering from diabetes mellitus.

In this demonstration our attention will be occupied by the consideration of 3 cases of diabetes in the juvenile form. Diabetes in youths and children, commonly termed "juvenile diabetes," differs from diabetes in adults very little except as to the etiologic factors and prognosis. Arteriosclerosis and the stress and strain of a strenuous life described as predisposing factors in diabetes in the adult obviously do not enter into the etiology of diabetes in children. The prognosis in children was con-

sidered most unfavorable and the expectancy of life was usually measured in months.

Modern treatment has been successful in prolonging the life of children and relieving them of many of the distressing symptoms.

The **etiology** of this disease in children is divided into predisposing causes and the exciting cause. Among predisposing causes heredity is a prominent factor, and 20 to 25 per cent. of the children give a history of the disease having occurred in the family. Heredity oftentimes exerts a favorable influence in the course of the disease. In patients with such a history it not infrequently happens that the symptoms are few in number and often are not progressive in character or even eventually may be cured in so far as the presence of symptoms. A history of a neuropathic make-up in one of the parents may be obtained.

Obesity is a predisposing factor in the history of children as frequently as it is in the history of adults.

Trauma, especially to the head or spinal column, has produced transitory and permanent glycosuria, and the course of disease, while often manifesting a tendency to be mild, differed in no way from diabetes ascribed to other causes. A history of familial diabetes is often found in these mild cases due to trauma.

A history of acute infections, as having immediately preceded the symptoms, can be obtained in a considerable number of instances. In the patients whose records we will have an opportunity of reviewing today 2 of the 3 patients give a history of an acute infection immediately preceding the onset of diabetic symptoms; in one the symptoms followed immediately an attack of influenza with pneumonia, and in the other a severe gastroenteritis preceded the appearance of symptoms of diabetes.

Just as in pneumonia, organs other than the lungs may suffer more severely and be permanently damaged by the products of the pneumococcus, so in a similar manner the pancreas (whose internal secretion has to do with sugar mobilization) may be the seat of an acute pancreatitis as part of a general infection. Thus may be explained the relation between diabetes and an acute infection.

It is also possible that diabetes or a diabetic tendency existed prior to the acute infection. If actual glycosuria had been present it was mild and symptomless, and the acute infection served to aggravate it to a marked and noticable degree in a manner similar to which a latent or incipient pulmonary tuberculosis is often precipitated into activity by acute infections, especially those involving the respiratory tract.

While heredity, obesity, trauma, dietary excesses, and nervous diseases are generally looked upon as predisposing causes, it appears that fundamentally the function of the internal secretion of the pancreas is at fault, which has to do with the utilization of carbohydrate in the body. Allen believes that in the normal individual the more carbohydrate ingested, the more is utilized by the body, whereas in the diabetic there exists a limit (threshold) of carbohydrate tolerance, which if exceeded results in glycosuria. This threshold is variable in different individuals and fluctuates under changed conditions.

Glycosuria may be the result of disordered function of one or more of the glands of internal secretion. It must not be forgotten that two of the principal storehouses of the carbohydrate of the body, the liver and skeletal muscles, must be reckoned as factors in producing faulty carbohydrate metabolism, should their function be disturbed through disease or injury.

The examination of the urine of a patient suspected of having diabetes, or of a known diabetic, should always be made upon a specimen of a twenty-four-hour collection. A single specimen taken may be negative for sugar, whereas a specimen taken two hours after a meal may show the presence of sugar. Usually the specific gravity of a urine containing sugar varies from 1.020 to 1.045, but a urine with a specific gravity as low as 1.005 may show sugar.

Urine which is to be tested for diacetic acid and acetone should be carefully kept in a cool place, otherwise the acid bodies which are volatile may not be present when tested for. A single fresh specimen is preferable for examination for these acid bodies. The percentage of sugar in the urine is not of great importance unless considered in connection with the

twenty-four-hour output. Specimens of urine taken at different periods of the day show different percentages of sugar. What is desired is the percentage of sugar in the twenty-four-hour quantity, so that the number of grams of sugar lost to the body by glycosuria may be estimated. The number of grams of sugar in a twenty-four-hour specimen of urine multiplied by 4 gives the number of calories of heat lost to the body by the glycosuria.

The percentage of sugar in the blood of normal individuals has been found to be in the neighborhood of 0.10 per cent. Diabetics if untreated show percentages varying from 0.10 to 0.40 per cent. Treatment will reduce the percentage of blood-sugar, and the nearer the percentage approaches the normal, the better the patient's progress can be considered. No glycosuria may be present, yet the percentage of blood-sugar may be high, indicating that unless the diet is reduced sugar will probably appear in the urine.

The **symptoms** in children do not differ essentially from those of adults, viz., polyuria, increased appetite, polydipsia, loss in weight and strength, and gastro-intestinal disturbances. The disposition of children is prominently and almost uniformly changed, and usually most trying to the parents. Irritability, fretfulness, sleeplessness, constant appeals for food and water characterize the average case of juvenile diabetes. Bed-wetting and itching about the external genitalia occur, and, together with the syrupy consistency of the urine with the deposit of sugar where drops of urine have evaporated, are early signs which may lead to the discovery of sugar in the urine. Diabetes in children has been described as having an acute onset, probably because of the brief duration of life in the past of the average case as compared with the course of the disease in an adult.

**Prognosis** in children has always been considered uniformly unfavorable, but with the advent of modern treatment much has been done to prolong life and keep the urine sugar free. Judging from the progress made in the past decade the future holds hope for further advancement along the line of treatment

leading probably to a cure rather than the arrest of the disease, with which most physicians are content at this time.

**Diagnosis.**—If the typical symptoms—namely, polyuria, polyphagia, polydipsia, and emaciation—are present the possibility of diabetes should suggest itself at once. In fact, with loss of weight and illness of any kind in which a diagnosis is not evident, the urine should be routinely examined to be sure that a glycosuria is not the underlying basis for the symptoms. Unquestionably as the frequency of urinary examination increases in children who are ill or in poor state of health, so will the discovery of glycosuria increase. We may consider patients who show glycosuria at intervals or in the presence of an acute infection as potential diabetics, and a régime should be adopted against the development of diabetes.

CASE I.—C. C. is an eleven-year-old boy who does not appear ill, as you see, but is rather underweight.

The family history is negative except for diabetes in the grandfather on the maternal side of the family.

The past history reveals measles, mumps, chicken-pox, and whooping-cough before the age of six. Birth normal, breast fed for one year. Mother always considered the boy normal. He is the only child, and except for the diseases enumerated above has always enjoyed the best of health; no abnormal appetite; weight has not been excessive at any time. No history of trauma. He has led an active outdoor life and has been a fair student. He is of a nervous temperament.

The present illness dates back to December, 1918, *i. e.*, months ago when he suffered an attack of influenza with pneumonia while at his home in Florida. The illness confined him to bed for three weeks, after which he was allowed to be up and about the house. He did not seem to gain strength or weight in spite of his excellent appetite and he has often had a rise in temperature. The physician attending him frequently examined his chest, but always pronounced it normal. He displayed no particular fondness for any particular kind of food. During the three months following the attack of influenzal pneumonia



he was confined to bed on four different occasions because he felt ill and ran a temperature varying from normal to 101.5° F. His diet was regular house diet with milk and eggs between meals, but in spite of the large quantities of food consumed he has been falling in health. At the end of three months following his initial illness his mother noticed the increased frequency of urination and the increased quantity of urine voided. It was also observed that he displayed an inordinate thirst and requested hot cakes and syrup as often as three times a day.

Four months after this attack of influenzal pneumonia, April 24, 1919, he arrived in Philadelphia after traveling on a train for forty-eight hours, living entirely on a diet consisting of large quantities of milk and eggs.

Our examination showed a very ill boy, drowsy, complaining of headache, respirations 32 per minute, temperature 98° F., with a definite odor of acetone about him. His bowels had not moved for forty-eight hours.

**Physical Examination.**—*Eyes*, negative; tongue dry, edges red, with reddish-brown coating on center of dorsum.

*Neck*—thyroid gland not enlarged, no glands palpable.

*Chest*—emaciated, expansion shallow, but equal, respiratory rate increased, with occasional long sighs.

*Lungs*—clear and resonant throughout. No râles are heard.

*Heart*—apex-beat, first sound lacks muscular tone. Rate regular, 80 per minute, and otherwise negative.

*Abdomen*—scaphoid, no tenderness or masses noted. Liver, spleen, and kidneys not palpable.

*Extremities*—negative except that the knee-jerks are exaggerated.

*Urine examination:* Single specimen shows specific gravity 1.032, acid in reaction, a cloud of albumin, numerous hyaline and granular casts, sugar 2 per cent., with diacetic acid and acetone tests strongly positive.

Treatment was begun to render his urine free of acid bodies as follows: 1 ounce of castor oil administered, followed in eight hours by a soapsuds enema. Water, as much as the patient would take of a glassful, was given the patient every half-hour,

and 4 ounces of water were introduced into the bowel every six hours.

Oatmeal gruel, orange water-ice, orange juice, bread, and the whites of 3 eggs in the form of albumen-water were given, and at the end of forty-eight hours the patient was voiding freely.

In the last twenty-four hours 1400 c.c. of urine were collected, with specific gravity 1.040, a trace of albumin with hyaline and granular casts, 5.6 per cent. of sugar, no diacetic acid or acetone. The patient was brighter, no hyperpnea or drowsiness present, but otherwise the boy's condition was practically the same as it was prior to coming to Philadelphia.

**Discussion.**—The hyperpnea, drowsiness, with acetone and diacetic acid present in the urine constitute a condition known as acidosis, which we would rather not meet in connection with diabetes. In examining a specimen of urine for acetone or diacetic acid it is necessary to examine a fresh specimen, otherwise these acid bodies, which are volatile, may be missed, especially if the urine has been kept in a warm room.

**DR. MOHLER:** Why did this boy develop acidosis on the train, or do you suppose the condition existed prior to boarding the train?

**STUDENT:** Was his urine examined for acetone or diacetic acid?

**DR. MOHLER:** There is no record of an examination for diacetic acid or acetone having been made, which should be done in every urine showing sugar, regardless of the clinical findings. This is most important and should not be neglected.

Let us suppose in the absence of a record of an examination for diacetic acid and acetone that none was present. What factor, if any, did his boarding the train have to do with his change in condition for the worse?

**STUDENT:** His diet while on the train if limited to milk and eggs may have produced acidosis.

**DR. MOHLER:** Yes, a patient accustomed to a diet rich in carbohydrate, if suddenly deprived of carbohydrate or diminished considerably, may, on a high fat protein diet, develop acidosis.

Is there any other point in the history favorable to development of acidosis?

STUDENT: The failure of the bowels to move for forty-eight hours would favor acidosis.

DR. MOHLER: Yes, failure to eliminate by means of free movement of the bowels will increase the tendency to the development of acidosis.

How is acidosis treated?

STUDENT: By increasing carbohydrate tolerance and keeping patient quiet and warm, giving alkalis, forcing fluids; if necessary give fluids by bowel, hypodermoclysis, and clearing thoroughly the intestinal tract.

DR. MOHLER: Prevention of acidosis is obviously preferable to treating the condition, and usually starvation or restriction of diet is immediately begun with the idea of clearing the urine of sugar. It would be dangerous to attempt to free the urine of this sugar by immediate starvation because the extreme grade of acidosis present demands prompt treatment before we can turn our efforts toward freeing the patient's urine of sugar.

Diabetics whose urines show strongly positive tests for diacetic acid and acetone are in danger, if not promptly and properly treated, of being precipitated into coma.

Our first efforts, then, must be to rid the patient of diacetic acid and acetone, and when this has been accomplished, treatment is directed toward rendering the urine sugar free. It is far better to have a large quantity of sugar in the urine of an untreated diabetic than to have a less amount of sugar with diacetic acid and acetone.

On April 30, 1919 patient was admitted to the hospital, considerable changed so far as his symptoms of acidosis were concerned, none being present, although the symptoms of diabetes continued very prominent. Ordinary house diet was given the patient during the first day in the hospital, and the twenty-four-hour specimen of urine showed a volume of 1500 c.c. of a specific gravity 1.040 with 110 grams of sugar in the urine. No diacetic acid or acetone was present. The chart shown on page 1317 gives an idea of the urinary output and sugar content during his stay in the hospital.

	April 30	May												
		1	2	3	4	5	6	7	8	9	10	11	12	13
Dates.....														
Quantity of urine in twenty-four hours, c.c.....	1500	2600	1600	1700	1700	2500	1800	1700	1000	1600	1900	1500	1800	1000
Specific gravity...	1.043	1.033	1.013	1.012	1.012	1.015	1.020	1.011	1.015	1.015	1.013	1.011	1.012	1.015
Sugar excreted in twenty-four hours, grams...	110	126	25	30	30	28	25	10	0	8	0	0	0	0
Acetone.....	positive	neg.	neg.	neg.	neg.	neg.	neg.	neg.	neg.	neg.	neg.	neg.	neg.	neg.
Diacetic acid.....	neg.	neg.	neg.	neg.	neg.	neg.	neg.	neg.	neg.	neg.	neg.	neg.	neg.	neg.
Weight, pounds ..	64	....	....	....	63½	62½	....	60	....	....	64	....	....	65
Blood-sugar.....	{ .12% <sup>1</sup> .13% <sup>2</sup>													

<sup>1</sup> Before breakfast.<sup>2</sup> One hour after noon meal.

You will notice that eight days were required to render the patient's urine sugar free, which was accomplished in the following manner: On the first day regular house diet was prescribed. The second day fats were omitted from the house diet, on the third day the protein in the diet was reduced one-half, on the fourth day, with one-half protein, the carbohydrate of the diet was reduced 15 grams (of which the house diet contains 80 grams), and each day approximately 10 grams less of carbohydrate were given, so that on the sixth day 35 grams of carbohydrate and 20 grams of protein were given in the diet. On the seventh day the patient received nothing but 6 ounces of hot clear broth every three hours, and a cup of weak tea in the morning and at night.

Our reason, as each of you know, for reducing the diet in the following order—the fat, then protein, then the carbohydrate—was because of the danger of producing acidosis in a child who just suffered from acidosis which was probably the preliminary stage of diabetic coma.

On the eighth day the patient became sugar free, his thirst and urinary output were notably lessened. On the ninth day, in addition to 6 ounces of broth every three hours and 10 grams of carbohydrate in the form of 5 per cent. vegetables, the patient drank milk taken from the tray of another patient, and as a result showed 8 grams of sugar in the twenty-four-hour specimen of urine.

The diet was increased 10 grams of carbohydrate each day in the form of 5 per cent. vegetables (a list of which you were given several weeks ago, and which appears in the standard textbooks on diet). At the end of the fourth day our patient was taking 40 grams of carbohydrate, on the third day 15 grams of protein (the whites of 2 large eggs) were added to the diet, and the protein thereafter was increased by 15 grams daily in the form of lean meat and chicken or fish, until 60 grams of protein were being taken. Butter and cream were now added to the diet and increased 5 grams daily until 45 grams of fat were being taken. The patient was allowed to leave the hospital on a diet composed of 40 grams of carbohydrates, 60 grams of protein, and 45 grams of fat for twenty-four hours, with instruc-

tions to save all urine. The patient's mother was taught to examine for sugar each morning.

One month later the patient weighed 68 pounds, 1 pound more than when he left the hospital and 4 more pounds than when he entered the hospital. His blood-sugar was 0.11 per cent. before breakfast. His diet was increased 10 grams each of carbohydrate and protein, and two months later 10 grams of fat were added to the diet.

On August 1st the patient was taking 50 grams of carbohydrate, 70 grams of protein, and 55 grams of fat.

Occasionally the morning examination revealed sugar, due to the boy taking diet other than that which had been prescribed for him.

The diet on November 1st was increased by 5 grams each of fat and carbohydrate, and the patient showed no sugar and the twenty-four-hour output of urine was 800 c.c., with specific gravity 1.021. No further blood examination has been made upon the patient because he has persistently refused to allow blood to be taken for sugar determination.

The boy, as you see him, in spite of his acidosis and glycosuria, seems to be in a good condition, and his mother states that he appears no different than before the attack of influenza, except that he is under weight by 12 pounds, as compared with his weight before he was taken ill. We are safe in predicting an uneventful course if his diet is carefully adhered to.

The patient is asked to report, personally or by letter, once a month, so that his case can be carefully followed.

It is important for the patient to know that we are interested in, first, that he does not gain weight too rapidly, for increase in weight means decrease in sugar tolerance, and second, that his diet is not increased in excess of that prescribed for him. The appearance of sugar in the specimen of urine examined is to be frowned upon, and the physician must let the patient distinctly understand that he is severely disappointed when he learns the urine shows sugar. The patient should be impressed with the importance of taking a partial fast day once a week, consisting largely of broth and 5 per cent. vegetables. We believe that

the prognosis in this patient will be very good if he adheres to the régime outlined for him, which we have every reason to believe will be done.

**CASE II.**—H. H. W., Jr., is a male child, age one year, ten and a half months on July 20, 1919.

**Family History.**—Patient is the first and only child of the parents. No history of renal, pulmonary, cardiac, or malignant disease, except an uncle of the mother of the patient suffered from diabetes. Parents both of about normal weight and in good health.

**Personal History and Present Illness.**—Normal birth, weight 8 pounds, breast fed, weaned at the end of one year. No gastro-intestinal disturbances until about eight months after the child had been weaned, then he suffered an attack of vomiting and diarrhea of about one week's duration. The child was very ill and lost considerable weight. Almost with the convalescence the parents noticed that the child began to have increased desire for food of all kinds, that increased thirst was complained of, with increased frequency of urination, and for the first time bed-wetting, a symptom which the child did not have prior to this illness. The stools were normal in number, but were usually green and pasty-like. No curds or masses of fat were ever noticed. The mother noticed that the urine was very thick in consistency and that it caused itching about the external genitalia. The urine was examined and contained 4 per cent. of sugar in a twenty-four-hour output of 950 c.c., with diacetic acid and acetone present. The carbohydrate was restricted and a high fat and protein diet was given, but the child was not doing well, and was brought to the hospital with the following report upon examination.

**Physical Examination.**—The patient is an emaciated male child, irritable, and crying almost the entire time during the examination.

*Head*—negative.

*Neck*—few palpable lymph-nodes.

*Tonsils*—apparently normal.



*Chest*—expansion equal, lungs negative.

*Heart*—pulse rapid (130 per minute), no murmurs detected.

*Abdomen*—distended with gas, no masses palpable, and apparently no tenderness.

*Extremities*—a diffuse pustular eruption over both lower extremities, which has been present for six weeks.

*Temperature*—100° F. Weight, 25 pounds.

The physical examination was very difficult, but apparently is negative except for the distention of abdomen and the pustular eruption.

The child was admitted to the hospital and the urine examination for the first twenty-four-hour period showed 600 c.c., specific gravity 1.026, with 4.8 per cent. of sugar, and positive test for diacetic acid and acetone. For a period of five days 50 grams of 5 per cent. vegetables (minced celery and spinach) with a tablespoonful of orange juice was given the child night and morning; 1 ounce of broth was given every two hours. The child was irritable and restless and constantly calling for food.

On the sixth day the fever rose to 104° F. A severe attack of diarrhea set in and the child was very sick, and for twenty-four hours it appeared as though he would not live. The child was dull and listless and refused all nourishment. Examination of the heart and the lungs was negative. Fluids, 1 ounce every two hours, were introduced by bowel. At the end of the fourth day of this acute gastro-intestinal attack the temperature dropped to 99° F. and the patient appeared brighter and took oatmeal gruel, 1 tablespoonful every two hours. This diet was kept up for two days, after which 50 grams of 5 per cent. vegetables were added, and the juice of one orange.

The diet was increased 5 grams of carbohydrate daily until the patient was taking 35 grams of carbohydrate in twenty-four hours. Protein was added in the form of white of egg (6 grams) after the urine of the child had been sugar free for twenty-four hours, and increased 3 grams daily until he was taking 35 grams. Fat was added in the form of cream.

	July										
	20	21	22	23	24	25	26	27	28	29	30
Dates.....											
Quantity of urine in twenty-four hours in c.c.....	600	700	700	450	300	350	300	400	300	400	350
Specific gravity.....	1.026	1.030	1.032	1.026	1.018	1.011	1.012	1.010	1.006	1.002	1.004
Percentage of sugar.....	4.8	2.8	4.4	3.2	1.2	none	none	none	none	none	none
Test for acetone.....	positive	neg.	neg.	neg.	neg.	neg.	positive	neg.	neg.	neg.	neg.
Test for diacetic acid.....	positive	neg.	neg.	neg.	neg.	neg.	neg.	positive	neg.	neg.	neg.
Albumin.....	faint trace	faint trace	faint trace	faint trace	faint trace	faint trace	faint trace	faint trace	faint trace	absent	absent
Granular casts.....	present	present	present	present	present	present	present	absent	absent	absent	absent
Weight, pounds.....	25	25	23	24	22	21	20	23	22	22	21 $\frac{3}{4}$

The child has progressed fairly well, except he often appeals for food, which is usually the case upon the day the diet is reduced to one-half the amount.

On December 1, 1919 the diet was as follows:

- 7 A. M. 1 tablespoonful of grape-fruit,  
3 tablespoonfuls of oatmeal,  
7 teaspoonfuls of cream (20 per cent.),  
3 ounces of broth.
- 10 A. M. 1 egg-white,  
7 tablespoonfuls of 5 per cent. vegetables,  
8 ounces of broth with 2 ounces of lime-water.
- 1 P. M. 1 ounce of lean meat, chicken, or fish,  
7 tablespoonfuls of 5 per cent. vegetables,  
8 ounces of broth with 2 ounces of lime-water.
- 4 P. M. 1 egg-white,  
7 tablespoonfuls of 5 per cent. vegetables,  
8 ounces of broth with 2 ounces of lime-water.
- 7 P. M. 1 tablespoonful of grape-fruit,  
1 egg,  
6 tablespoonfuls of vegetables,  
8 ounces of broth with 2 ounces of lime-water.

The patient weighed  $22\frac{1}{2}$  pounds and is stronger than at any time since the beginning of his diabetes.

**Discussion.**—The case illustrates satisfactory results due to the faithful mother with an unlimited amount of patience. To care for a child who is constantly in search of something to eat is a task that is far from easy for those who must care for a child with this disease. On December 1st the mother stated in her letter that the boy, although he has not gained any in weight, seems brighter and freer from the symptoms than at any time since the onset of the disease. The same procedure is followed in the treatment of acidosis in children as in older patients, and the diet consists of practically the same, except in the case of vegetables, which should be thoroughly minced so as to cause no gastro-intestinal disturbance.

Passive exercise in the form of daily rubs of olive oil, massaging the entire body, has helped the boy to utilize his diet.

Within five days after admission to the hospital the pustular eruption on his lower limbs disappeared and has been absent ever since.

It will be noticed that the casts and albumin disappeared after the urine became sugar free.

The disappearance of albumin and casts in 2 cases of diabetes in adults after the urine has become sugar free has been observed. The irritating action of the sugar on the kidneys may have had to do with the presence of albumin and casts.

It is important to restrict the diet in diabetics during an acute infection because the sugar tolerance is temporarily lowered, and if the diet is not restricted, even in so slight a condition as acute rhinitis, we have noted the presence of sugar upon a diet of the same composition under which sugar was repeatedly absent.

The prognosis of this child does not appear as favorable as Case I, but each month that the child's urine remains sugar free must encourage us to make our prognosis more favorable.

**CASE III.**—K. A. is a male child aged two years and eight months, when he first came under our observation October 16, 1917.

**Family History.**—Hebrew ancestry, with no history of diabetes on maternal or paternal side of family. First and only child of the parents.

**Personal History.**—Birth normal, breast fed for eighteen months. He had been a fat baby and was considered normal in every way by the parents until his present illness. His appetite was normal. Patient has had measles and chicken-pox. No history of trauma obtained.

**Present Illness.**—Seven weeks ago for the first time the patient's mother noticed that the child was urinating very frequently and rather larger quantities than heretofore. The child also had a fever. The fever subsided in the course of three or four days, but, as the mother recalls, the child began to ask for food of all kinds and became very irritable. The child had

been weighed one month before he became ill and now weighed 4 pounds less than at that time, his present weight being 31 pounds. Very prominent symptoms are the child's restlessness, irritability, and more or less constant fretfulness.

**Physical Examination.**—Pulse 86 per minute; temperature 98° F.; respirations 24 per minute. *Head*—negative. *Tonsils*—appear normal. *Neck*—no palpable glands. *Chest*—negative. *Heart*—negative. *Abdomen*—no masses or areas of tenderness. *Extremities and skin*—negative.

A single specimen of urine examined showed a specific gravity of 1.030, sugar 4 per cent., with diacetic acid and acetone present. The family physician placed the child upon a diet composed largely of protein and fat and restricted the carbohydrate. The child's urine was at no time sugar free during the past seven weeks. The child on October 16, 1917, according to our notes, came under our observation at the hospital, and a twenty-four-hour specimen of urine shows 1000 c.c. containing 3.1 per cent. of sugar with diacetic acid and acetone. The blood-sugar before breakfast was 0.17 per cent. and 0.22 per cent. two hours after the noonday meal. The patient is not yet under dietetic treatment, the regular soft-diet tray having been ordered for him.

The patient, because of the brief duration of symptoms, appeared to be one most favorable for the prompt restriction of diet to broth and orange-juice, in order to render his urine free of sugar, diacetic acid, and acetone.

Realizing the difficulty of instituting a period of what amounts virtually to starvation it was decided to take the child on a forty-eight-hour auto trip, so that he would not see any food except his broth and orange-juice; 6 ounces of broth were given every three hours and a tablespoonful of orange-juice night and morning. The amusement afforded by the trip, together with the fact that the child neither saw nor smelled food other than his own, served to lighten the task of diet restriction.

At the end of the second twenty-four-hour period the urine amounted to 500 c.c. and upon examination showed no sugar, diacetic acid, or acetone; the child was apparently none the

worse for the starvation. His weight was  $\frac{1}{2}$  pound less than before the fast was begun.

According to various observers a child at the age of two years requires approximately 80 calories for each kilogram of weight during a twenty-four-hour period, and an attempt was made to provide a diet for this child that would keep the urine sugar, diacetic acid, and acetone free, and provide the largest number of calories. During the third twenty-four-hour period the determination of carbohydrate tolerance was begun by the feeding of 5 per cent. vegetables in the form of cabbage, spinach, and lettuce leaves.

The child was less irritable, yet the task of restricting the diet was a most difficult one. The vegetables were thrice boiled, thereby lessening the amount of carbohydrate taken and increasing the bulk. The danger of causing gastro-intestinal disturbances was lessened by passing the vegetables through a flour sieve, and no intestinal upset was encountered; 150 grams of 5 per cent. vegetables, when thrice boiled, were calculated as having but 3 per cent. of carbohydrate or 5 grams of carbohydrate, and each day 5 grams of carbohydrate were added until sugar appeared at the end of the fourth day, when the carbohydrate was reduced to 15 grams. At the end of forty-eight hours albumin-water was given to the child, giving the white of one egg (6 grams of protein) in a pint of water during the day, and this was increased by 3 grams daily until 21 grams were given at the end of seven days from the beginning of the period of tolerance determination. Fat was then supplied to the child in the form of 20 per cent. cream, each ounce of which contained approximately 6 grams of fat, 1 gram of carbohydrate, and 1 gram of protein. Due allowance for protein and carbohydrate of cream was made on total carbohydrate and protein allowance.

The diet of the child was approximately:

15 grams of carbohydrate.....	60 calories
21 grams of protein.....	84 calories
20 grams of fat.....	180 calories
Total.....	324 calories

The patient on this diet became brighter and his weight remained about 25 pounds for one year. The greatest patience and vigilance were necessary, and not infrequently during the past two years has the child shown sugar by taking unnoticed, usually, bread, crackers, or jam. His urine has been collected daily, and a specimen of the twenty-four-hour collection of urine has been examined by his father for sugar, diacetic acid, and acetone, the latter two constituents at no time having been present.

The diet during November, 1919 has been as follows:

30 grams of carbohydrate.....	120 calories
40 grams of protein.....	160 calories
30 grams of fat.....	270 calories
Total.....	550 calories

in the form of 5 per cent. vegetables, meat, eggs, fish, oysters, and broth.

Weight, 30 pounds. Blood-sugar 0.12 per cent. (fasting).

Usually one day a week has been observed as vegetable day—a diet consisting of 200 grams of 5 per cent. vegetables, with a tablespoonful of orange-juice.

The child, as you see him, is bright, active, intelligent, and has grown to know that he cannot indulge in exercises and eat food that other children can. Although underweight, he has a fair amount of endurance. Twelve hours out of every twenty-four are spent in sleep. In addition to the exercise of walking the patient receives daily one-half hour general massage with olive oil.

This patient has endured the disease for more than two years, and although he has passed through no acute illness, the prognosis seems very favorable. Every month or six weeks an examination for blood-sugar is made, since it appears that the blood-sugar percentage must be kept at 0.12 per cent. or, still better, 0.10 per cent. If the blood-sugar is kept within the limits of normal the diet must be kept low, which will necessarily keep the weight of the patient low.

These case histories and patients represent what can be done



and how accomplished with children suffering from diabetes mellitus. Not all children yield so uniformly to treatment as these patients have done.

In the 3 cases considered it was possible in every instance by restricting diet to render the patients sugar free and keep them sugar free and slowly increase the quantities of food tolerance. The caloric values of the diets have been kept low, and by keeping the fat content of diet low as compared with fat content of adults we not only prevent sugar from appearing in the urine but also do away with acidosis, and finally increase the tolerance for carbohydrate, protein, and fat.

FROM THE MEDICAL CLINIC OF THE UNIVERSITY  
OF PENNSYLVANIA

**The Treatment of Valvular Heart Disease Before Failure of  
Compensation**

By ALFRED STENGEL, M. D.

**Edema of the Lungs**

By DAVID RIESMAN, M. D.

**Meningitis**

By H. R. M. LANDIS, M. D.

**Syphilitic Aortitis**

By GEORGE WILLIAM NORRIS, M. D.

**Three Instructive Cases**

By JOHN H. MUSSER, JR., M. D.

**Mumps**

By JOSEPH SAILER, M. D.

**Significance of Heart Murmurs in Young Individuals**

By EDWARD H. GOODMAN, M. D.

**Hodgkin's Disease with Jaundice as an Early Symptom**

By O. H. PERRY PEPPER, M. D.

**The Treatment of Catarrhal Jaundice**

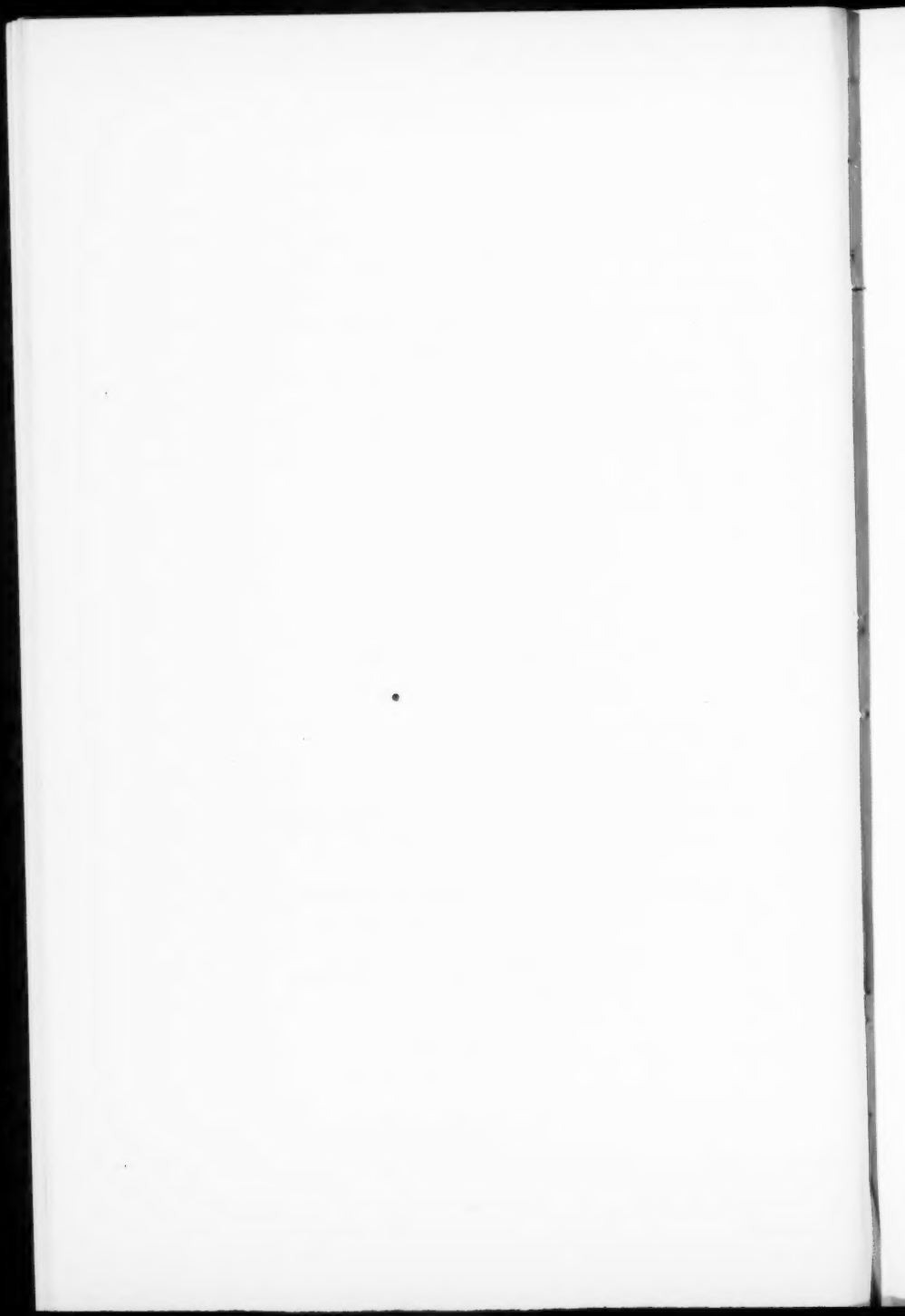
By ARTHUR H. HOPKINS, M. D.

**Drug Inebriety**

By JOSEPH C. DOANE, M. D.

**Painless Gastric Crises**

By CLIFFORD B. FARR, M. D.



## CLINIC OF DR. ALFRED STENGEL

UNIVERSITY HOSPITAL

---

### THE TREATMENT OF VALVULAR HEART DISEASE BEFORE FAILURE OF COMPENSATION

DR. STENGEL: Gentlemen, I propose to show you 2 cases of valvular heart disease, and then to discuss with you the treatment of this condition before there has been any evidence of failure of cardiac power, or, as it is commonly expressed, before there is failure of compensation. First, I shall ask Mr. A. to report to us the history and his physical examination of the first case.

MR. A.: Mrs. S., aged twenty-eight, was admitted to the hospital complaining of some palpitation and forceful beating of her heart on exertion.

*History of Present Illness.*—The patient was well until four years ago, when, shortly after her marriage, she first noticed palpitation of her heart on exertion or excitement. This has continued until the present time, and during the last four months has become more or less troublesome. Occasionally she suffers from an irritative, unproductive cough, and she has noticed that this is more marked when she is tired or nervous. She thinks that she has always been of a nervous temperament, but has become more so during the last few months. She is decidedly apprehensive regarding her symptoms. Her digestion and gastro-intestinal system generally are normal. Lately she has lost a little weight.

*Past Medical History.*—At the age of seven she had a severe attack of diphtheria which was followed by "heart trouble," which continued for some months after recovery from the diphtheria. At fourteen years she had muscular rheumatism, which lasted for more than three months. During this time

some recurrence of heart trouble was noted, and subsequently for several years she was weak and unable to be as active as girls of her age. She has had four or five subsequent attacks of diphtheria, the last one several years ago, and has had antitoxin on each occasion. The tonsils were operated on at ten years. The operation was incomplete and a second tonsillectomy was performed two years ago. Menstrual history negative.

*Social History.*—Household duties; meals at regular times. Coffee, 2 or 3 cups a day. Exercise rather restricted on account of her cardiac condition.

*Family History.*—Married four years ago; no children; no miscarriages.

*Physical Examination.*—The patient is a rather healthy looking young woman, twenty-six years old, color decidedly good; cheeks and lips red; general physique normal. General inspection negative. The heart is slightly enlarged to the left, the left border being 10 cm. to the left of the midsternal line. The right border is 3 cm. to the right of the midsternal line. The apex impulse of the heart is strong. There is no palpable thrill. On auscultation a distinct systolic murmur is heard at the apex and transmitted somewhat toward the axilla and back. The first sound of the heart is rather sharp, and there is at times a suggestion of a presystolic murmur, but this was not constantly heard. The second sound of the heart at the pulmonary area is moderately accentuated and is sharper in this situation than over the arch of the aorta. Examination of the lungs is entirely negative. The abdomen is not distended; liver and spleen are not palpable. The patient is rather nervous in manner and the reflexes are moderately accentuated. Blood and urine examinations are entirely negative. Blood-pressure: Systolic, 146; diastolic, 70. Pulse-rate 100; pulse regular and of moderate volume.

DR. STENGEL: What diagnosis have you to offer in this case?

MR. A.: Mitral valvular disease.

DR. STENGEL: To what do you attribute this lesion?

MR. A.: Possibly to the severe attack of diphtheria or to rheumatic infection.

DR. STENGEL: Is diphtheria one of the infections which you recognize as frequently productive of endocardial lesions and subsequent valvular disease?

MR. A.: My understanding is that diphtheria is less likely to produce endocarditis than some other acute infections, like rheumatism, scarlatina, and septic infections, but I believe it is capable of producing valvular disease. Moreover, this patient is reported to have had several attacks of diphtheria, and perhaps the infection has been more serious in her case than in others.

DR. STENGEL: I think your statement is correct so far as the relative importance of diphtheria and the other infections is concerned, and in this particular case one may doubt whether the repeated attacks reported as diphtheria were truly diphtheria. The patient evidently has had a bad condition of her tonsils which eventually required their removal; and it is a notable fact that in such cases there are often recurring attacks of acute tonsillitis, sometimes with membrane and possibly most frequently occasioned by streptococcic infection. I may go farther in stating that such tonsillar conditions are more likely to be followed by endocarditis and valvular disease than diphtheria, which is capable of producing endocarditis in a certain proportion of cases, but does not seem to do so in a very large number. We must remember that a diagnosis of diphtheria is frequently made upon the discovery of a membranous deposit which may be due to other organisms than the Klebs-Löffler bacillus.

An added fact of some significance in the story is the record of a rather prolonged rheumatic attack, called muscular rheumatism, which occurred after the primary, so-called, diphtheria, and during which her heart symptoms are alleged to have recurred and perhaps increased. This attack suggests that her tonsillar infections from the beginning may have been rather of the type allied to rheumatism than diphtheria. Leaving the question of etiology, let me ask you what sort of mitral lesion you believe to be present?

MR. A.: Mitral stenosis with regurgitation.

DR. STENGEL: And upon what do you base your opinion?

MR. A.: Upon the fact that there is a sharply accentuated first heart sound and a suggestion of presystolic murmur, which would point in the direction of mitral stenosis, and upon the existence of a systolic murmur which suggests mitral regurgitation.

DR. STENGEL: Is there anything else to suggest mitral stenosis here?

MR. A.: I understand that the left ventricle is less apt to be enlarged in stenosis than in cases in which mitral regurgitation is the pronounced lesion.

DR. STENGEL: I think that is correct, and moreover, if you will study the statistical tables you will find that mitral stenosis is a more common occurrence in the female than in the male sex. I should like to call your attention also to a certain physiognomy that you will discover quite frequently in cases of mitral stenosis, and which is clearly indicated in our patient. I have constantly observed that these young women with mitral stenosis present a quite strikingly characteristic appearance in their rather delicate features, their high color, often with a somewhat cyanosed appearance of the lips; and a generally not unhealthy appearance. Having the history pointing to a cardiac trouble and this appearance in a young woman, one cannot infrequently, make a fair guess that mitral stenosis is present. I do not wish to emphasize this too much, but the appearance of our patient has suggested it to my mind, and I thought it worth while mentioning in passing.

Now, Mr. A., will you tell us whether you consider the cardiac condition here as one in a state of compensation?

MR. A.: I hardly know how to answer the question, because my understanding is that when compensation is broken, patients suffer with dyspnea, cyanosis, dropsy, and such symptoms, indicative of the failure of circulation, and these symptoms are not present in our patient. Still the patient has had some cardiac disturbance.

DR. STENGEL: There is, however, something in her history about shortness of breath on exertion or excitement.

MR. A.: Yes, there is; though I do not know that the amount of shortness of breath was more than a person in ill health from any cause might have under those conditions.

DR. STENGEL: There is, in addition, some history of palpitation and a sense of forceful action of the heart, is there not?

MR. A.: Yes; but I do not know whether these can be regarded as indications of disturbance of compensation.

DR. STENGEL: Your answer brings up a very important matter regarding cases of mitral stenosis, and I should like to place before you my own views on this situation. The evidence of broken compensation is ordinarily sought in some disturbance of the mass movement of blood, such as would be indicated by shortness of breath resulting from congestion of the lungs, enlargement of the liver, peripheral cyanosis, edema, and the like, and, according to this mode of interpretation our patient could hardly be regarded as one with disordered compensation. But in my experience cases of mitral stenosis frequently give evidence of the beginning of cardiac deficiency by manifesting disturbance of cardiac action considerably before they suffer from any disturbance of the mass movement of the blood. They contrast strongly with other forms of cardiac disease, with the possible exception of cases of rheumatic aortic regurgitation, where something of the same sort occurs. It is not infrequent in my experience to find a rather protracted history of palpitation and subjective sensations of disturbed heart action before any evidences of circulatory derangement make their appearance. I regard these symptoms as evidences of the beginnings of decompensation, though, of course, they are not expressive of such derangement of the movement of the blood as one finds at a later stage. From this point of view it would, therefore, appear that the case we have before us is one of mitral valvular disease with the beginning of decompensation. Before we go further with the discussion let us bring in the second case.

MR. A.: Case II.—Mr. R. S. B., aged forty, was admitted on account of a slight chill, followed by fever.

*History of Present Illness.*—The patient was in normal health



until three days ago, when, after an exposure to cold, he had a chill followed by fever and general aching in his limbs. His temperature rose to  $102\frac{3}{8}^{\circ}$  F.; his pulse was 100, and respirations 22 on admission. Examination then showed a slight redness of his pharynx and tonsils and a little filmy deposit in his throat. There were no other symptoms of consequence.

*Past Medical History.*—The patient has always been healthy so far as he knew, having suffered from no severe illness either in childhood or up to the present time. He occasionally had slight acid indigestion and required laxatives from time to time. At seventeen years he had typhoid fever quite severely, and he has had moderate attacks of sore throat or tonsillitis, but apparently no severe attacks. Never has had rheumatism.

*Family History.*—Negative.

*Social History.*—Has always been engaged in outdoor work, frequently rather severe, but without known injury to himself. Habits good. Married; two children.

*Physical Examination.*—The patient is a sturdy looking man, face a little flushed, lips rather red. External inspection negative. Heart distinctly enlarged to the left, extending 11 plus cm. to the left of the midsternal line, and 4 cm. to the right. The apex impulse is forceful and there is a loud systolic murmur at the apex transmitted to the axilla and back. The first sound of the heart is considerably obscured by the murmur. The second sound at the pulmonary area is accentuated.

Blood-pressure: Systolic, 120; diastolic, 70. Pulse-rate 94.

There is still slight redness of the tonsils and pharynx, but this has diminished since his admission three days ago. His temperature has now fallen to normal.

DR. STENGEL: Did you find any evidence of pulmonary abnormalities?

MR. A.: No; the percussion sounds over the lungs were entirely normal, and there was no change in the breath sounds nor any râles.

DR. STENGEL: What did you find on examination of the abdomen?

MR. A.: Nothing abnormal. The liver and spleen were not

palpable, and the abdominal condition generally seemed entirely negative.

DR. STENGEL: What is your diagnosis in this case?

MR. A.: Mitral valvular disease.

DR. STENGEL: And what type of lesion do you believe to be present here?

MR. A.: Mitral regurgitation.

DR. STENGEL: Do you regard this as the result of a preceding endocarditis, or the result of some change in the muscle of the heart, with possibly dilatation of its chambers?

MR. A.: I believe it is a valvular lesion due to a preceding endocarditis, because there is no preceding history of muscular weakness and no evidence that there has been progressive decompensation which might result from a muscular disease and occasion dilatation with relative incompetency of the mitral valve. The enlargement of the heart might perhaps point in the direction of a dilatation, but this could also be the result of a rather long-standing valvular lesion, such as mitral regurgitation.

DR. STENGEL: What have you to say, then, as to the compensation at the present time?

MR. A.: So far as I can see there is no evidence whatever that the patient presents any decompensation. His symptoms have been entirely negative so far as his circulation is concerned, excepting that since the onset of the acute illness, which brought him to the hospital, he has had a little rapidity of the heart action, but no more than one would find in cases of fever of this grade in normal individuals.

DR. STENGEL: Will you state the findings on which you base the opinion of a mitral regurgitation?

MR. A.: The enlargement of the heart both to the left and right, the presence of a distinct systolic murmur, partially replacing the first heart sound and transmitted clearly toward the axilla and back, and the accentuated second pulmonary sound.

DR. STENGEL: Do you feel that the absence of any history of rheumatism or other acute infection likely to produce an endocarditis would militate against your diagnosis?

MR. A.: I think that the diagnosis would be more certain if there were a history of rheumatism or some other infection, but I believe that it is quite common to find cardiac lesions without any such preceding history. Minor infections of the throat and diseases early in life that did not appear of great gravity at the time might occasion cardiac lesions that would remain unrecognized until later in life.

DR. STENGEL: I think your answer to this is correct. May I ask you now as to the possibility that some other explanation than a valvular lesion might account for the apical systolic murmur?

MR. A.: I remember that we have been taught to regard murmurs at the apex or over the body of the heart as of less definite significance in younger persons, particularly when there is no cardiac enlargement, but, in this case the hypertrophy or enlargement both to right and left, combined with the murmur, seems to me to warrant the diagnosis.

DR. STENGEL: Without going into a long discussion of this question of the diagnosis of mitral regurgitation, I merely wish to emphasize the importance of our recognizing the frequency of systolic murmurs that cannot be regarded as significant of a mitral valve lesion. You may recall my pointing out to you in a previous clinic how frequently such murmurs can be developed in young persons by cardiac overaction when there is no suspicion of a valvular defect, and in many cases the murmur may even be quite constant. Unless the evidences of cardiac enlargement are quite clear and some related history indicates the probability of a cardiac lesion, we should hesitate in concluding that a systolic murmur signifies a damaged mitral valve, but I do not wish to pursue this subject at the present time. Let me now ask Mr. A. what he regards the pathologic lesion of the mitral valve to be?

MR. A.: Chronic valvulitis or endocarditis.

DR. STENGEL: Do you mean by this a progressive disease in the mitral valve?

MR. A.: No; I suppose that there is no active disease present at all.

DR. STENGEL: Then do you regard the term "chronic endocarditis" as entirely justified?

MR. A.: I do not know, but the term "chronic endocarditis" is generally used to designate such valvular lesions.

DR. STENGEL: I agree that you are entirely correct in your statement, though I wish to emphasize the importance of our observing that there is not in this condition a continuing process. As a matter of fact, in a large part of the cases of what we call chronic endocarditis we deal with the after-result of a disease process long since completed, and now a matter of past history. The valve has been damaged by an ancient acute endocarditis which has ceased and has left scar tissue as its result. There is, therefore, nothing in the valvular defect that truly represents a chronic disease any more than there is a chronic disease present in any scar-tissue formation. It is important to bear this in mind because we must have clear conceptions with regard to valvular disease with reference to its continuance and extension; or, on the other hand, its remaining a stationary condition. In the case of syphilitic valvular disease we have, of course, a different state of affairs. Here there is truly a chronic valvular infection which tends to extend for some time, perhaps for an indefinite period of time. In the other case a rheumatic endocarditis leaves behind it a valvular defect which, once established, does not of necessity increase beyond the possible contraction of the scar tissue formed and perhaps the deposition of calcareous salts.

Now, Mr. A., let me ask you how you understand compensation to be established in cases of valvular disease?

MR. A.: I believe that the usual explanation is that the heart muscle increases in power so that it may exert more force in its contractions and thus overcome the valvular defect.

DR. STENGEL: I think we can accept this explanation as, roughly speaking, adequate, with the addition that there are changes in the size of the cavities of the heart which bring about an adjustment of the circulatory difficulties occasioned by the valvular disease. These, with the increased power of the heart, enable it to perform its part of the circulation despite the exist-

ence of a valvular derangement. Now, Mr. A., what do you understand by reserve power in the heart?

MR. A.: Reserve power is the power which every normal heart possesses beyond its ordinary needs and available for emergencies.

DR. STENGEL: How does the reserve power of a compensated heart in valvular disease compare with that of a normal heart?

MR. A.: There is never as great a margin in the cases with valvular disease, the reserve power sometimes being very little.

DR. STENGEL: Then I understand that the compensated heart may be in danger of failure because its inadequate reserve power may be exhausted?

MR. A.: Yes, I believe it is commonly thought that the reserve power in a compensated heart may be readily exhausted.

DR. STENGEL: Now, gentlemen, let me take up the discussion of these two cases from this point, and let me first of all direct your attention to some matters relating to the general circulation, but of importance to the heart itself, that have a bearing on the management of such cases as these which have been presented to you.

Please note that we have here two instances of valvular disease of the heart: one, apparently, mitral stenosis; the other, mitral regurgitation, in which the compensation has not been seriously disturbed in the former and not at all in the latter. The important question of treatment of such cases as these, and this, of course, applies to the great majority of the cases of valvular disease of the heart at some stage of their clinical history, is to determine the measures which shall be pursued to safeguard the heart against subsequent decompensation. While you may not find it stated in so many words in medical text-books or in clinical discussions, there seems to be a prevailing opinion that valvular disease once established somehow tends eventually to cardiac breakdown through gradually increasing myocardial weakening and subsequent failure. Here and there medical writers have taken a different view of the situation, and to quote only one, I recall that Romberg says, "Hypertrophy, as such, does not carry with it the germs of later cardiac weakness through

gradual exhaustion or the like." If you will recall what was stated a few moments ago, that the valvular lesion, as such, does not in most instances tend to be progressive, but remains fixed as it was left by the healed endocarditis, and as the compensation for this valvular lesion is established *after* the lesion has been created, there is nothing in the circumstances which must inevitably occasion cardiac breakdown. Why then do cases of valvular disease so commonly end in decompensation? A brief answer to this might be formulated as follows: The reserve power of the heart which protects the normal heart against the well-nigh unavoidable strains of daily life is lacking to a very considerable extent in cases of valvular disease, which, therefore, more readily yield to such recurring strains. Unusual safeguards must, therefore, be provided for the individual having a cardiac lesion.

Now, what are the conditions which affect the cardiac power unfavorably? To answer this question we must direct our thoughts to the circulation as a whole rather than to the heart itself, and must study the various influences that are useful in maintaining the normal blood flow. Several years ago I published a discussion of this subject under the title "Extracardiac Causes of Failure of Compensation in Valvular Diseases of the Heart,"<sup>1</sup> but did not specially enlarge upon the deductions that could be drawn from this discussion in the way of the proper management of such diseases.

First let us review very briefly the facts regarding the circulation. The heart, as the central organ of the circulation, is the most important factor in maintaining the blood flow, but exercises its power almost wholly on the arterial side. By successive systolic contractions it maintains a grade of pressure in the arterial system that insures a steady flow of blood into the arterioles and capillaries. Within this area three factors are of fundamental importance: (1) The power of the heart, (2) the proper elasticity and caliber of the vessels, and (3) the efficiency of the vasomotor mechanism. The last of these factors may be variously influenced by toxic

<sup>1</sup> Amer. Jour. Med. Sci.

(renal, intestinal), nervous (cerebral), or secretory (endocrine gland) influences. Any unfavorable condition within the arterial and capillary area rendering the onward movement of the blood more difficult must, of course, put a strain upon the heart, and must, therefore, be taken seriously into account in the management of cases of cardiac diseases.

Beyond the capillaries—in the return circulation through the veins to the heart—the force of the cardiac action plays no important part in the movement of the blood, for the pressure within the arteries is dissipated in overcoming the resistance within the arterioles and capillaries, and is reduced to but a few millimeters of mercury in the beginning veins. From this point onward to the heart other agencies become effective. In the first place, muscular contractions compressing the veins drive the blood on toward the heart on account of the valvular arrangements in the veins, and physical activity is on this account of the utmost importance to the circulation. Second, the inspiratory expansions of the thorax by suddenly lowering the pressure in the great veins near the heart exert a suction effect upon the blood and draw it from the periphery toward the heart; third, the movements of the diaphragm and abdominal walls by compressing the abdominal veins force the blood upward toward the thorax; and fourth, gravity to a small extent aids in the return flow from the head. Should unfavorable conditions interfere with any or all of these factors, there must follow stagnation of blood in the venous channels, and in consequence eventually disturbance of the heart itself by reason of its receiving an insufficient supply of blood. Thus in cases of serious muscular weakness (as after illnesses) or of muscular inactivity from any cause venous stasis in the extremities is frequently observed. Again, in cases of markedly varicose veins considerable proportions of the total mass of blood may be stagnated in the dilated veins. In cases of thoracic diseases interfering with expansion (large effusions, aneurysms, tumors, chronic emphysema, kyphoscoliosis, muscular atrophy) the loss of aspiratory power may seriously disturb the venous blood flow, and in each of the conditions named circulatory fail-



ure may be largely the result of this cause. In similar conditions and also in cases of enteroptosis, or of primarily weakened abdominal walls or paralysis of the diaphragm, intra-abdominal stasis may be a disturbing factor capable of causing circulatory failure. Please note that none of these conditions directly exercise an unfavorable influence on the heart to any appreciable degree, but indirectly, by depriving it of the due amount of blood, interfere with its proper action. In general terms we may state the proposition that anything which causes accumulation of blood in the peripheral veins or in the splanchnic area must exercise a deleterious effect upon the heart.

Another group of circumstances from which may proceed the causes of cardiac breakdown are composed of nutritional disorders and infections. The purely local (gastro-intestinal) and mechanical disadvantages of overfeeding may be passed over for the moment, to be discussed later in connection with the consideration of treatment, but I wish at this place to refer to the possible direct cardiac strain that may be occasioned by superalimentation. Sir Clifford Allbutt has been an earnest advocate of the view that overfeeding affects the circulation injuriously by increasing the viscosity of the blood or otherwise altering it so as to overload the heart. I do not believe it will be profitable to discuss *in extenso* this aspect of the overfeeding question, but it is well you should know that some regard it as important.

The rôle of subsequent infections in bringing about cardiac decompensation is far more important. Whatever the immediate cause of decompensation, we recognize that myocardial disease is always a fundamental condition. To a certain extent every case of endocarditis is accompanied by myocardial disease from the beginning, but manifestly in well compensated cases the degree of myocardial trouble is not sufficient to render the heart incompetent. Such hearts, however, are doubtless more vulnerable to further infective weakening than normal organs, and thus infections of all sorts, whether mild or severe, have an especial significance in patients having valvular lesions. Thoracic infections are perhaps of particular danger because the cough-



ing, disturbances in breathing, and interference with the pulmonary circulation incident to them adds a purely mechanical factor of cardiac strain to the common danger of infective weakening of the heart muscle. Some writers have stated a belief that gall-bladder infections especially endanger the heart muscle, but I am inclined to believe this opinion is not entirely accurate. There can be no doubt of the fact that myocardial disease frequently results from chronic gall-bladder infections, but it seems to me the explanation of the apparently marked tendency of such infections to occasion myocardial disease is to be found in the fact that such chronic infections of the gall-bladder so frequently occur at a time of life when myocardial disease is imminent on account of the age of the patient rather than because of any special tendency of these infections to attack the heart muscle.

#### TREATMENT IN COMPENSATED VALVULAR HEART DISEASE

DR. STENGEL: Let us now consider the treatment which should be prescribed for cases of valvular heart disease without evidences of decompensation. What treatment do you think the patients you have shown require?

MR. A.: I think such cases are better off without treatment.

DR. STENGEL: Undoubtedly that is correct so far as the usual forms of drug treatment are concerned, particularly the administration of digitalis. It is hardly necessary to say that nowadays few doctors fall into the error of prescribing digitalis or other cardiac stimulants merely because a murmur or other evidence of valvular disease, but without decompensation, has been discovered. It has sometimes been suggested that small, so-called tonic, doses of digitalis may aid in strengthening a not altogether competent heart, but I shall not attempt to discuss this question now. The claim is at best doubtful. There can be no question, however, of the impropriety of administering full doses of digitalis in compensated cases. In such, cardiac disturbance is almost certain to result.

The question then arises regarding drugs in general. Is anything in the way of medicaments called for?

Mr. A.: General tonics might be useful.

DR. STENGEL: Yes, provided there is need of them, which does not always appear to be the case, and can scarcely be thought to be true of the 2 cases we have seen.

Not infrequently, however, in cases of valvular disease in young patients—mainly mitral cases—there is a certain degree of anemia, and in these instances slight but repeated disturbances of compensation frequently recur and threaten more complete decompensation. I shall not attempt now to explain fully the reasons for such anemic conditions. Suffice to say it may be due to associated diseases, local or general, to the existence of a local infection, as, for example, diseased tonsils, to persistence, in an inactive form, of the endocardial infection, or to reinfection of the valves or myocardium. Whatever the cause of the anemia, I can testify to the usefulness of iron medication in such cases. Some of these patients seem, indeed, to have a remarkable tolerance (and perhaps need) of iron. The tendency to recurrence of decompensation to which they had been notably liable before may disappear after the free administration of iron. The best preparation by far is a freshly made Bland's capsule—modified by using equal amounts of dried sulphate of iron and bicarbonate of soda—the latter replacing the potassium carbonate of the official combination. With iron, general tonics containing strychnin, nux vomica, and quinin are undoubtedly of use.

Of course many other conditions requiring treatment may arise in cases of compensated heart disease, but we are not now concerned with accidental occurrences.

What other treatment is to be suggested to your patients?

Mr. A.: Nothing more than that they should be careful in their mode of life.

DR. STENGEL: This answer I fear all too accurately repeats the prevailing attitude of the medical profession. As far as it goes, no objection can be made to it. The same advice, of course, would be given to any patient seeking advice regarding a chronic disease, but it gives the patient no real information.

You will note as I continue the discussion why I took so

much time and pains to detail the aids to the circulation that are found outside the heart itself. We are under the obligation of helping a partially crippled heart to perform its necessary work with the least strain, and of safeguarding it against any unfavorable influences from without. We are confessedly unable to add to its power or to remove the lesion that is present, but we can lighten its labor and protect it from certain kinds of harm. This requires that we advise the patient in detail regarding his mode of life and occupation, his diet, exercise, clothing, etc.

Before entering on the particulars of this discussion let me say a few words regarding the danger of making the patient unduly nervous or apprehensive by revealing to him the existence of a cardiac defect and by indicating the proper safeguards. Many physicians are so fearful of the effect on the patient's mind that they habitually practice concealment, if not equivocation. Nothing could be more harmful to the patient's best interests. *Remember, however, first, that you are correct in your diagnosis, and second, that you properly gage your patient in choosing the language of your explanations and advice.* The cases in which I have seen harm done by plain speaking have usually been either cases in which the assumed cardiac disease did not exist, and a nervous disorder of heart action was increased by the patient's alarm, or cases in which an overstatement or untactful statement of the conditions present was made. A quiet and simple explanation of the actual state of affairs will rarely, if ever, do any lasting harm; it may be a momentary shock, but when we remember that the whole future of the patient is involved in his giving intelligent co-operation, the risk must be taken even if it be far greater than I believe it to be. I am not unmindful here of the fact that valvular disease is very frequently diagnosed in young people on account of the presence of a systolic murmur or slight cardiac arrhythmia when no valvular or even muscular disease of the heart exists; but you must remember the first condition I gave above as a necessary precedent to your venturing on a frank discussion with the patient or his friends. Truth telling as a practice necessarily implies that the truth be known. Where uncertainty exists, silence is golden.

The results of concealment of the existence of a cardiac disease and of failure to advise patients properly is daily seen in the wards of our hospitals. Dr. Hoehn has recently tabulated for me our recent records of cases of valvular disease admitted to the hospital in a condition of more or less advanced decompensation, and has noted especially the occupations of these patients just before the final break occurred. Of 337 cases, 82 are recorded as engaged in what we set down as "hard work," and this number does not include many of the female patients who are listed as having been employed in "housework"—an occupation that assuredly is often anything but light. We cannot, of course, know how many of these patients were advised regarding their cardiac condition, and it is but fair to believe that many would have disregarded advice to seek different employment; but it is altogether probable that a large proportion had no knowledge of their real condition.

Let us now review systematically the plan of life that is most desirable for the cases under discussion.

**Exercise and Play.**—Many of our patients come under observation in childhood, and we are therefore primarily concerned with the matter of exercise and play rather than work or "occupation." The same fundamental principles are, however, applicable in either case. I have already pointed out the advantages of muscular contractions in aiding the return circulation, but have not considered the direct effects of exercise on the work of the heart. It is a familiar fact that all forms of muscular exertion increase the rate of the heart action, and physiologic observations show that the blood-pressure is usually elevated. Exercise is, therefore, to a certain extent always primarily a strain on the cardiac power, though this may by suitable regulation be sufficiently slight to be overbalanced on the favorable side by the useful effect of the muscular activity in aiding the flow of blood in the veins. Moreover, as the blood-supply of muscles increases during exercises, by dilation of the vessels, it is probable, as some investigations seem to show, that moderate exercise may ultimately have the effect of lowering peripheral pressure and actually decreasing the work of the heart. Whether

this be true or not there can be little or no doubt that the beneficial effects outweigh any harmful effects in the case of moderate physical exercises. Aside from such direct results we have also to remember that there are indirect results, such as a greater supply of fresh air, improved pulmonary expansion, better digestion, healthier action of the skin, all of which count on the side of improvement of the circulation. In the practical management of the play of children with cardiac disease there are, however, difficulties that are sometimes hard to overcome, for it is usually impossible to regulate the amount of exercise as we should like. The common effort to prevent such children from taking any part in the games of their friends probably results from the feeling that it is impossible to control the amount of exercise and to prevent excesses rather than from the thought that all exercise should be forbidden. The result is that the little patients are kept in a state of constant repression and are eventually got into a state of nervous apprehension while they are at the same time deprived the moderate exercise that would certainly be beneficial. The dangers of occasional excess have been, I think, greatly exaggerated, and my own practice has been rather to encourage than discourage play. The only embargo that seems to me necessary is that on the rougher games, and in school children or adolescents on competitive sports of all kinds.

After adult years have been reached we must, of course, rely upon the voluntary co-operation of our patients, and then it seems to me essential to explain the conditions fully to the patient—pointing out the advantages of moderate exercise on the one hand and the dangers of severe strains on the other. As a result of a rather large experience in this type of disease I feel justified in saying that I believe more harm is done by efforts at repression than by giving a fairly free rein to our youthful patients. This should not, however, be interpreted as a justification for careless physical examinations on the part of those who have to do with the selection of boys or girls who are to make up athletic teams. I happen to have examined a number of boys and young men who have been members of football, hockey and basket-ball teams, and rowing crews despite the fact

that they had rather serious valvular lesions. This, of course, indicates a lax system of physical examination.

I wish to add one other thought which perhaps hardly needs mention. This is that exercise, to be most beneficial and least apt to be harmful, even if occasionally excessive, should be frequent and regular rather than occasional.

Special forms of exercise may be advisable for certain cases on account of associated conditions that interfere with the circulation and thus put an undue strain upon the heart. Thus in cases having poor chest development or defective thoracic musculature, pulmonary exercises, calisthenics, etc., may be highly beneficial, particularly in youthful patients who have not attained their full development. To secure better thoracic expansion some of this same group will require removal of adenoids or tonsillectomy, for, as you know, mouth-breathers are notoriously liable to chest deformities as a result of improper expansion. Incidentally, let me add that the removal of diseased tonsils or adenoids is of further value in disposing of possible sources of reinfection of the heart.

**Occupation.**—Two important factors in the matter of suitable occupation are that severe and sudden muscular strains should be avoided and that excessive fatigue on account of necessarily long hours or close application should be prohibited. The belief that only sedentary occupations should be allowed cardiac cases is practically in the class with the plan of forbidding children with such diseases all forms of play. An absolutely sedentary life would doubtless do more harm than one of moderate excess in work. Milder forms of manual labor with a reasonable amount of fresh air will prove more satisfactory than indoor work without exercise of any kind. The selection of an occupation, of course, depends upon a great number of factors aside from the medical desirability, but fortunately the list from which it can be made is a considerable one. Heavy manual labor is, of course, to be excluded, and also lighter work in which the manual operations are so frequent that there is a constant strain and at the end of the day a serious degree of fatigue. Work exposing the individual to extremes of temperature and, still worse, to

rapid changes of temperature is also objectionable. Occupations involving long or constant standing are manifestly undesirable.

In general, employments which permit of moving about partly in the open air or in well-ventilated workrooms and occasionally of resting, which involve some manual work of a light sort and which do not require long hours, are most suitable.

**Rest and Sleep.**—I may add to what has just been said regarding work that sometimes even a definitely unsuitable occupation may be more tolerable by the institution of rest periods. In general, while the cardiac case should seek employment in which he will have a certain amount of physical exercise, it is important that he shall have more than the average requirement of rest. Frequently you will find that persons with compensated heart disease seem to need a greater amount of sleep than normal people. Instead of discouraging this tendency and of attempting to conform to ordinary standards such individuals should be advised to rest an hour or two longer than is the ordinary custom. Sometimes a practice of resting an hour at the end of the day's work is found an advantage by those who are affected with great fatigability. The hour's rest tranquilizes the circulation and restores the patient so that his appetite for the evening meal and his digestion are greatly improved. The homely rule of "early to bed and early to rise" may well be adopted without reservation or amendment of the former part; the latter part need not be too rigidly observed.

**Diet.**—The proper dietary regulations may be epitomized as: Simplicity, variety, and moderation, but the greatest of these is moderation. The bearing of diet on the well-being of cardiac cases is shown in various ways. In the first place it is a commonplace of daily observation that overfilling of the stomach, especially when accompanied by flatulent distention, may prove disastrous, especially in elderly patients. Second, constant overfeeding tends to cause intestinal disorders with fermentative or putrefactive decomposition of the intestinal contents and consequent systemic toxemia. A whole train of unfavorable results may follow this condition, including disturbances of the vasomotor mechanism, myocardial and arterial diseases, renal



troubles, etc. Third, the habit of overeating tends to cause constant overfilling of the splanchnic blood-vessels and thus indirectly disturbs the general circulation and the heart action. Finally, the increased fatness of overfed persons causes cardiac strain by reason of the excessive weight of the individual and more especially by reason of the dragging of the overloaded omentum and abdominal walls upon the diaphragm and other intra-abdominal structures. You will find as a matter of practical experience that patients who are moderate, careful eaters preserve their compensation far better than those cursed with a vigorous appetite and a disposition to indulge it. Doubtless, as I said a while since in speaking of Sir Clifford Allbutt's views, other factors than the mere mechanical effects of fatness are involved. There may be conditions of the blood in such persons that tend to raise arterial tension and put an injurious strain on the heart. Certain it is that some of the cases of "essential arterial hypertension" that we encounter in practice present no other discoverable explanation for their condition than that of constant overeating.

I am tempted to make a digression here to call your attention to the relation of stature to cardiac competency. You will find that large framed and excessively tall people are far more prone to early decompensation than those of lesser dimensions. Such oversized patients must particularly beware of overfeeding and excessive weight. One recalls the dictum of Napoleon that 5 feet, 4 inches is the best size for a soldier. Unfortunately, we cannot select a suitable height for our patients, but we can emphasise the importance of not adding weight to other handicaps. Sometimes when undesirable fatness and abdominal girth have been acquired it will be found especially advantageous to direct the wearing of an abdominal support, a plan which I have repeatedly found of the greatest value to persons with beginning evidences of cardiac overstrain.

To resume the discussion of diet, let me remind you of the plan devised many years ago by Oertel for the treatment of persons suffering from "fat-heart." His plan consisted essentially in the reduction of the fluids of the diet, and is



a valuable method for this class of patients. It is, however, useful in other types of cardiac disease, such as valvular disease in persons not unduly stout. Undoubtedly the ingestion of large amounts of liquid causes cardiac strain without contributing any counterbalancing advantages, though some patients fall into the habit of excessive water drinking in the belief that the flushing out of the system will overcome the effect of accumulations of waste products. As a matter of fact such persons usually begin by being overeaters, thus primarily overtaxing the circulation, and end by drinking excessive quantities of water which adds to their cardiac difficulties. Whatever we may conclude as to the desirability of enjoining a distinctly dry diet, either habitually or now and then, there can be no doubt that the constant habit of drinking large quantities of fluid is undesirable for those who have cardiac lesions.

Stout patients may with advantage be ordered an occasional Karell diet. This, as you will remember, is a diet consisting of a restricted amount of milk—say, 1000 c.c. per day—and no other food. In decompensated cases with some edema this plan of diet continued for short periods of time has a diuretic effect and is in other ways useful; but its employment is frequently advantageous in perfectly compensated but overfat individuals with cardiac disease. We must remember that the tissues in obesity contain a larger amount of fluid than those of normal people, a proof of which is seen in the rather rapid loss of weight in the beginning of various "cures" for fatness when the treatment involves some restriction in the amount of fluid ingested. A judicious alternation of the Oertel and Karell principles of dieting is undoubtedly desirable for overfat cardiac cases.

**Care of the Digestive Tract.**—Aside from the matter of diet the cardiac defective needs to exercise as great care in all that pertains to improving digestion and the normal action of the bowels as the chronic dyspeptic. Regularity in meals and prevention of occasional excesses, thorough mastication, and avoidance of hurry and excitement at mealtime are important conditions. As for the movements of the bowels, the habit of regularity should be established early in life, or if this has not been the case, and

cannot be accomplished later, mild laxative measures, such as the use of suppositories, enemas, mineral oil, saline waters, etc., should be resorted to. It is particularly important for cardiac cases that habitual constipation with its resulting straining be avoided. In cases of beginning decompensation you will often find that occasional portal depletion by the use of stronger saline or other laxatives—calomel, jalap, and the like—is useful treatment.

**Clothing.**—Suitable clothing is of the utmost importance. Cardiac cases are highly susceptible to the effects of chilling, and the resulting minor infections are peculiarly dangerous in their consequences. Without going into detail as to clothing, let me simply suggest one or two important matters. Warmth of the extremities in particular and avoidance of dampness are especially necessary; and the wearing of a woolen abdominal band is often advisable. Unnecessarily heavy clothing should be avoided, and especially such as contributes more weight than warmth.

**Bathing.**—The habit of cool or cold bathing is highly desirable for persons with somewhat sluggish circulation, such as we find in many of the cases of cardiac disease even when compensation seems quite adequate. This condition is especially marked in cases of mitral valve disease, and is often entirely wanting in cases of aortic disease. Indeed, cases of aortic regurgitation when well compensated are more likely to have an overactive peripheral circulation owing to the dilated condition of the arterioles, and suffer from warmth and flushing rather than coldness. In mitral cases, on the contrary, coldness of the hands and feet, especially in the morning and when the patient is fatigued, and the habit of "sleeping cold" (getting chilled in the early morning hours and requiring additional blankets or covers) are frequently the cause of discomfort. In such patients a momentary plunge in cool or even cold water followed by friction with a coarse towel acts as a powerful stimulus to the circulation and at once gives the patient a sense of warmth. Some persons find it difficult to muster up courage for such a morning plunge, but may find it possible to stand or kneel in warm water and sponge the head and chest and douche the back with colder water. Eventually they may grow

accustomed to a cool dip. Such a bath should always be very brief—not above a half minute or minute—and should be advised only to youthful patients, though it may be continued to middle life or later once the habit is formed. Prolonged cold bathing is, of course, depressing and therefore highly objectionable in such cases.

A hot bath (not warm or lukewarm) is equally stimulating to the circulation, and may in some cases be substituted; but as the relaxed skin is more sensitive to cold after the bath, hot bathing (whether as a tonic or for cleanliness) should be allowed only at times of the day when the patient can remain indoors after it.

Salt baths and salt rubs are undoubtedly useful in certain cardiac cases, but, generally speaking, have their proper place only in the treatment of later stages—when compensation has begun to fail. They may, of course, be used earlier, but it is not in my opinion wise to advise measures that suggest invalidism until the necessities of the case warrants them.

**Climate and Altitude.**—Much has been written regarding the effects of climate and altitude in cardiac disease, and the subject might be drawn out to great length. I wish only to direct your thoughts to one or two important considerations.

First, in the matter of altitude, you will find, I think, that the prevalent idea that the sooner the patient can be gotten to sea level the more satisfactory will be his condition does not work out in experience. Moderate altitudes in fairly equable, though perhaps somewhat bracing climates, are more likely to be helpful to cardiac cases than sea level, and particularly the sea level in warm regions where there is a certain deadness of air that proves depressing. In general, my experience has been that anything up to 1000 feet is desirable. It is, of course, admitted that higher altitudes are distinctly disadvantageous, notwithstanding the fact that among the residents of such elevated regions are found the usual proportions of cardiac cases, and these do not appear to suffer more severely than the same type of cases at lower levels. A closer study of the results in such original inhabitants of high altitudes is desirable and might reverse the statement I have just made. There is no doubt, however, of the truth of the

statement that cardiac cases previously residents of low altitudes are affected injuriously by going to greater elevations.

Second, changeable, damp climates are undesirable because of the tendency to repeated "colds" which are particularly dangerous to cardiac cases in whom a bronchial infection is always more apt to be prolonged than in other people, and, with its accompanying cough, adds to the cardiac strain and seriously endangers the compensation. Moreover, each repeated infection, however mild, exposes the individual to the possibilities of added myocardial degeneration.

**Treatment of Infections and Other Diseases in Cardiac Cases.**

—The occurrence of an infection must always be regarded more seriously in cardiac cases than in the normal individual, and the patient should be put to bed, no matter how mild the infection may seem. There is always the danger of reinfection of the heart, which, of course, may more readily follow a neglected infection than one cut short by proper management. Local conditions, such as diseased tonsils or adenoids, pyorrhea, and dental root infections, must be treated as possible sources of mischief. One must not, however, become extreme in dealing with such troubles, and I especially protest against the disposition occasionally encountered on the part of some who with more zeal than discretion alarm their patients by overstating the danger of cardiac reinfection, and make this the basis for possibly needless tonsillectomies, tooth extractions, and the like.

It would, of course, be a highly desirable thing if we could outline prophylactic measures that would safeguard our cardiac patients from the possibility of all sorts of infection; but we should place our reliance on the general plan of hygienic life such as I have attempted to outline for you. For the rest, our dependence must be placed upon careful management once an infection has begun, and upon a suitably prolonged rest in bed after convalescence is established.

Bronchial infections are attended with an additional risk to the heart on account of the straining and sudden elevation of blood-pressure that results from coughing. In such cases it is

desirable to use sedatives to control cough rather more freely than in persons without cardiac disease.

Gastro-intestinal diseases in which severe constipation or marked flatulent distention of the stomach or bowels occur should be managed with particular care to obviate these unfavorable conditions as speedily and completely as may be possible. The dietary treatment of these cases must be directed with especial care.

Conditions affecting the peripheral circulation such as result from vasomotor disorders caused by nervous derangements and general systemic weakness, or from varicose conditions of the veins, may occasion serious cardiac disturbance. The group of cases in which vasomotor disturbances are met with is closely related to the type of cardiac weakness variously designated as weak heart, cardio-asthenia, etc., and I wish particularly to emphasize that this condition may occur in individuals who have no real organic cardiac disease, but also, and more seriously, in those already handicapped by a valvular defect. In either case systematic and graduated exercise, massage, salt rubbing, and other hydrotherapeutic measures may be required.

The effect of varicose veins of the legs in cardiac cases is often very decided, especially in persons beyond middle life. In these cases the wearing of elastic stockings or correction of the difficulty by surgical means may help materially in the restoration of the circulation when medical means alone will not avail.

## CLINIC OF DR. DAVID RIESMAN

PHILADELPHIA GENERAL HOSPITAL

---

### EDEMA OF THE LUNGS

THIS subject was suggested to me by your President, Dr. Watson, and in many ways it is one of the best choices of a general topic that could be made.

You will be surprised if you look up the standard text-books to find how little space is devoted to pulmonary edema. One of the most recent and "thickest" books on diseases of the heart and lungs contains hardly more than a page or two on this important subject. I have wondered why it is being treated in such a step-motherly fashion. I do not know. Anyone who sees a good deal of clinical medicine cannot help but have a deep interest in edema of the lungs, and those of us who passed through the recent influenza epidemic have acquired a very profound respect, if not an actual awe, for it.

What do we mean by pulmonary edema? By pulmonary edema we mean the presence of serum in the air vesicles. In mild cases it is confined to the air vesicles, but in severe cases it may collect in the finer bronchial tubes and the air passages in general, and may transude into the interstices of the pulmonary tissue.

In order to understand the subject of pulmonary edema we ought, I think, to classify the types of edema, and when we begin to do that we realize that some forms of edema are unimportant and, from the standpoint of practice, negligible. Now it is to those that the majority of text-books give attention; they devote so little space to the subject because those varieties do not require much consideration. But there are other forms of pulmonary edema, the importance of which is hardly exceeded

by any other condition that we have to deal with in the thorax. That is a strong statement, and yet I am going to prove to you that this is a fact.

I am in the habit of classifying pulmonary edema as follows:

1. **Passive Edema.**—That is the common edema of heart disease, of valvular and muscular disease of the heart with decompensation. When we say "decompensation" the average person thinks of the fully developed form; just as when we speak of cirrhosis of the liver or of tabes dorsalis, the beginner at least thinks of the classical type of these diseases. Now, the classical type of most diseases are so well known that he who runs may read; it is the imperfectly developed, the incipient cases of any chronic affection that do not diagnose themselves and have to be diagnosed.

The meaning of that is this: We can often diagnose the oncoming of cardiac decompensation before decompensation is manifest, and the thing that usually tells us is pulmonary edema. In many cases, before any swelling of the feet, any shortness of breath, any enlargement of the liver, any effusion into serous cavities appears, a little crackling can be heard at the base of the lungs. That is pulmonary edema. It is the earliest stage of the passive variety. You might say, Is that not passive congestion? Many do not distinguish between passive congestion and edema, but there is a distinction. A patient with disease of the heart of any duration always has some congestion of the lungs, but he does not have pulmonary edema, the leaking of serum out of the vessels, until decompensation begins to ensue. Passive congestion, as such, may be considered to exist when in valvular or muscular heart disease there is a loud accentuated second pulmonic sound and enlargement of the heart's dulness to the right, indicating an overfilling auricle; an overfilled auricle means an overfilled lung, and that is a congested lung which does not become an edematous lung until some of the serum oozes out of the engorged vessels into the air cells.

Now I have often found in cardiac cases—coming for other reasons—a little edema, shown by râles at the bases. I tell the patient that he must go to bed for a few days, otherwise he is



likely to have shortness of breath and swelling of the legs; perhaps even more serious symptoms. The edema being an early sign, enables us, if we detect it, to begin treatment of decompensation in its incipency. The value of this is self-evident.

2. Then, again, we find passive pulmonary edema in elderly persons, or in any person who has been long lying on his back. This is sometimes spoken of as hypostatic edema. It is an advanced stage of hypostatic congestion—a **gravity edema**. It has a peculiar feature. It is what the French call a **reversible edema**, changing from side to side as the patient is turned. I have just come from another hospital where I found such a shifting edema in an old man. The edema is a passive-congestion edema; it is chiefly significant in affording a good soil for bronchitis and pneumonia in the aged. A similar edema, explicable on the same grounds, is found in typhoid fever, in septicemia, in severe influenza, and in a protracted fever from any cause. In the senile edema, if there is no contravening circumstance, it is advisable to have the patient sit up as early as possible, as that helps to get rid of the edema.

3. Another form of pulmonary edema is that due to Bright's disease—**nephritic edema**. It is most common in acute and chronic glomerular and tubular nephritis.

The question may come to your mind, Is nephritic edema a passive edema, an edema of the same nature as that of heart disease? I should have to answer the question by saying: "It does not appear to be so; it is questionable." In some cases of chronic Bright's disease of the so-called interstitial variety there is often some edema due to cardiac failure; but I do not think that edema of the legs or of the abdomen, or of the face in the other, the so-called parenchymatous nephritis, can be explained on a cardiac basis. Some of the forms of nephritic edema are due to a toxic irritation of the blood-vessel walls, making them more permeable, or to changes in the blood, chiefly an increased sodium chlorid content. In any event, the edema of Bright's disease is not a purely mechanical one.

4. The next form I wish to speak of is what might be called **localized inflammatory edema** of the lung. This is important



from the diagnostic point of view on account of its presence in the earliest stages of lobar and influenzal pneumonia. It gives rise to those fine crepitant râles that are so characteristic of oncoming consolidation.

An acute edema of general distribution is seen in gas poisoning. It has long been known in the industries to be a consequence of the inhalation of irritant vapors. In the late war gas poisoning, as you know, was very common. The gases, mostly of an irritant character, distribute themselves in the lungs and cause an acute inflammatory edema, which is not confined to the air vesicles, but involves other portions of the lung as well. I shall not go into the details of this topic, but would refer you to a valuable paper published by Dr. G. W. Norris, of the University of Pennsylvania, in the *Journal of the American Medical Association*.

**5. Acute Terminal Edema.**—In pneumonia, influenza, septicemia, and a variety of other acute diseases one frequently finds a generalized edema as a serious complication. Indeed, death often seems to be the direct consequence of a rapid filling up of the lungs with edematous fluid. During the influenza epidemic pulmonary edema was a frequent and much dreaded occurrence, against which we were often powerless. When we come to speak of treatment I shall point out the measures that sometimes prove successful in combating the edema. An acute edema of a similar character has been described as occurring in acute rheumatic fever. Personally, I have never seen it.

An analogous edema plays a part in postoperative shock. The cause of such edema—which often deserves the name “agonal”—is to be found in toxic myocardial weakness and in vasomotor paralysis.

**6. Postcritical Edema.**—This is an edema that is found in the pneumonic lung after the crisis. The fever is gone; the patient seems perfectly well, and wants to get up; but if one listens over the lung one hears a little residual crackling which often persists for a long time. Many patients with such an edema do well if allowed to sit up, but they should not be allowed to walk about or to go out of doors.

In some cases of influenzal bronchopneumonia an edema of large and small râles, affecting apices as well as bases, persists for a long time after the temperature has reached normal. It is accompanied by marked prostration, a slight degree of cyanosis, and a tendency to sweating. Atropin, digitalis and dry cupping have seemed to influence it favorably. Regarding the etiology, it is difficult to make any definite statements; the association with profuse perspiration suggests a vasomotor factor.

7. I now come to speak of an edema which is perhaps more important than any other. For years it has greatly interested me. I have called it **acute apoplectic edema**, and because of the marked tendency to recur it has also been designated **acute recurrent edema**. I can best describe it by reading to you brief histories of a few cases that have come under my observation (Riesman, Amer. Jour. Med. Sci., January, 1907).

CASE I.—Mrs. L., aged forty-six years; numerous attacks of acute edema coming on without apparent cause. First attack in 1902, two days after death of mother; lasted one and a half hours. No albuminuria in the beginning, later small amount of albumin, urine pale, low specific gravity, casts from time to time. Attacks characterized by dyspnea, rattling breathing, mucous râles, sense of fear and terror, high tension pulse. Patient neurotic and subject to laughing and crying spells. Death occurred December, 1905, in an attack lasting ten minutes, during which frothy, bloody fluid gushed from mouth and nose. Autopsy: Chronic interstitial nephritis, hypertrophy of the heart; intense pulmonary edema, fresh petechial hemorrhages in the pleura and pericardium.

CASE II.—Mrs. McC., aged forty years; asthma of several years' standing. During the past winter and spring repeated attacks of intense dyspnea with rattling breathing, frothy expectoration, bubbling râles, alarming collapse, with pallor, feeble pulse, clammy skin. Attacks entirely different from asthmatic paroxysms, but occurring during asthmatic periods. A number of attacks within two or three months. None since going south.

CASE III.—Mrs. N. S., widow, aged fifty-eight years. For three or four years attacks of anginoid pain in precordium, with radiation into left arm. Attacks always associated with orthopnea and signs of pulmonary edema; the latter at times confined to left lung, generally bilateral. Best results during attacks from dry cupping and nitroglycerin. Death from cardiac dropsy; no autopsy.

CASE IV.—I have another case among my records that occurred during pregnancy. The patient, suffering from albuminuria, was in such an alarming state as the result of the edema that Dr. Montgomery who saw her with me thought that labor should be induced. Under ethyl chlorid an attempt was made to stretch the cervix, but it could not be done. A piece of gauze was introduced, free bleeding followed, but labor did not take place. The woman nevertheless improved and soon recovered and the urine cleared up. Six weeks later the edema recurred. I promptly bled her from the elbow, with good results. She was afterward delivered without difficulty and has had no other attacks since.

Permit me to recite one other case:

CASE V.—Mr. W., aged seventy-two years; always healthy. Seized suddenly one night with intense edema of the lungs; chest filled with moist râles; reddish, frothy fluid gushing from mouth; dyspnea, collapse, unconsciousness, paralysis of right arm and leg, pulse irregular, reduplication of first sound at apex. Marked arteriosclerosis, urine (catheter) albuminous. Disappearance of pulmonary signs in two or three days, palsy lasting a little longer; eventual recovery. Another attack a month later, January 23, 1904, not so severe; recovery. Numerous others, usually with stupor or coma and variable degrees of fleeting palsy and aphasia. Last attack March 19, 1904, at 3 A. M.; intense dyspnea, unconsciousness, fluid gushing from mouth and nose, paralysis of right arm; partial restoration of consciousness following venesection. Death; no autopsy.

This form of edema, about which very little has been written,

is, as you see, very important. Three of the patients died and two nearly died. It may occur under a variety of conditions. One of my early cases occurred in a patient with mitral stenosis. When I came to the house the doctor in attendance said the man was practically dead. True, he seemed to be in articulo mortis; the breathing was rattling and pitifully laboring; the chest full of râles; bloody froth was gushing from nose and mouth; the pulse at the wrist was imperceptible. To our amazement, the man reacted, was soon himself again, and lived for several years afterward.

In pregnant women having mitral stenosis this form of edema is quite common. It is, of course, true that many women with mitral stenosis pass through labor unscathed, but the possibility of an acute fatal edema of the lungs hangs constantly over them.

The same type of edema occurs in chronic myocarditis. Six days ago a physician brought to my office a man forty-eight years old, complaining of most distressing attacks coming on chiefly at night, in which he would get extremely short of breath and very anxious, and would expectorate large quantities of frothy fluid. I asked him what the color of this fluid was, and he answered, "I think it was pinkish." I said, "You think? Do you not know?" He replied, "To tell the truth, doctor, I am totally color blind." Since he thought it was pinkish it must have had some color to suggest pink to him. Within a year he has had about ten attacks, each lasting from one to two hours. At the beginning of an attack he gets hot and perspires; afterward he feels cold and subsequently falls asleep. His blood-pressure in my office was 170 systolic, 80 diastolic, the pulse 148. Because of a prominence of the eyes and a rapid pulse my assistant got the impression that the man had exophthalmic goiter, but upon examination I found extreme irregularity and rapidity of the heart, there being 168 beats over the heart and 148 at the wrist. There was no goiter. The heart was enlarged and a loud blowing mitral systolic murmur was present. The case was one of myocarditis with auricular fibrillation.

A short time ago I saw a man fifty-seven years of age who said he had heart trouble. He suffered from attacks in

which he said he choked and choked until the lungs were empty. During such a seizure he expectorates from a half-gallon to a gallon of bloody fluid, getting relief only when all the fluid is up. He is an intelligent man, has been carefully observed by his physician, and has been seen by nearly every local consultant and by everyone of note within a considerable radius of Philadelphia. Altogether he has had from thirty to forty mild and about six severe attacks. He knows when they are coming on; he gets "heated up and very short of breath." "It is like a fright; I have a choking sensation, and a pain in the right chest crossing over to the left. I have to sit up or choke to death." The attacks come on usually during the afternoon, but they may occur at night. The longest lasted an hour and a half. There is no provocative cause apparent, but he himself says that he has neurasthenia. I am quite sure we should all become neurasthenic in similar circumstances. His blood-pressure is not very high, but there is distinct evidence of myocarditis.

I have seen the same form of edema also in association with angina pectoris. One of the patients whose history I read to you had anginoid pains in the precordia.

Next to myocarditis, the most frequent associations of acute apoplectic edema are with hypertensive arteriosclerosis and with chronic nephritis. The most fulminant cases I have seen were in patients with arteriosclerosis and high blood-pressure.

Barring angina pectoris, nothing in the way of an acute attack is so alarming as this form of pulmonary edema. It puts life in peril just as much as angina pectoris, yet columns have been written about the latter and scarcely anything about the former. A striking feature is its *tendency to recurrence*. Thus the one man of whom I spoke had had thirty or forty minor attacks and six or seven severe attacks, in every one of which he thought he would choke to death. On looking through the literature I found the record of a woman of forty-five years who had had seventy-two attacks within two years (Lissaman, *Lancet*, February 8, 1902).

The pathogenesis of recurrent pulmonary edema is not definitely known. Most of the experimental work of any value

dates back some years, so that there is here a great field for research by the newer methods. Dr. Welch, of Baltimore, working in Cohnheim's Laboratory, found that pulmonary edema was dependent upon a disproportionate action of the two ventricles. If the left gets weak while the right is still strong, the latter continues to pump blood into the lungs which cannot get out. The lung, in consequence, becomes overfilled and the blood-serum transudes from the capillaries into the air vesicles. This theory, while one most widely accepted, is not entirely satisfactory. In the first place it does not explain the cause of the sudden difference in the behavior of the two ventricles, and does not explain the acute development within the course of a few minutes of an edema menacing life. There is probably another factor, perhaps a vasomotor disturbance. In saying this I am well aware of the fact that the question of a vasomotor supply to the pulmonary vessels is still under discussion. While Bradford and Dean (*Jour. of Physiol.*, 16, 34, 1894) contend that the pulmonary arteries possess vasoconstrictor fibers, Brodie and Dixon (*Ibid.*, 30, 476, 1904) have come to an opposite conclusion. The question is an open one, but certainly there is a good deal in clinical observation to strengthen belief in the existence of a pulmonary vasomotor mechanism.

In reading the histories of cases I have practically given you the symptoms of recurrent edema which, please remember, is as distinct a syndrome as angina pectoris. To make the picture clear I will, however, bring the symptoms once more before you. They are sudden, intense dyspnea or orthopnea, deep cyanosis, air-hunger, rattling breathing, râles all over the chest—often most abundant at the apices—and fear of impending death. There is usually a large amount of thin, frothy sputum; in bad cases it may reach enormous quantities, gushing from mouth and nose and drowning the patient in his own juices. The fluid is frequently blood tinged, looking like raspberry juice. The existence of such expectoration is, however, not necessary to enable a diagnosis of pulmonary edema to be made. There are cases in which nothing is brought up, but in which the diagnosis is easily made by the other symptoms and

the presence of moist râles all over the chest. Sometimes there is, in addition to the pulmonary edema (and this proves that the condition is not a purely local one), a temporary aphasia or a trivial palsy; stupor or unconsciousness may also occur, showing that the brain has suffered. In many cases an extreme vascular distention is present.

The prognosis of apoplectic pulmonary edema is like that of angina pectoris. Who can give a reliable prognosis in that disease? In pulmonary edema a man may be brought to the brink of the grave, as close as I have never seen happen in any other condition, and yet survive. The prognosis is very much governed by one's cast of mind, whether one is an optimist or a pessimist. An optimist believes that the patient will weather the attack; a pessimist, that he will not. Prognostically I am an optimist.

Perhaps I ought to say a word about the diagnosis—what can acute recurrent edema be mistaken for? In one of the cases of which I have spoken, my doctor friend had made a diagnosis of cardiac asthma, because he was unfamiliar with the true condition. Charcot once said: "An obscure disease is often diagnosed by merely thinking about it." Not only must we distinguish it from cardiac asthma but also from other forms of asthma and from angina pectoris, more particularly when there is no expectoration. As I have stated, angina pectoris and acute pulmonary edema may occur together.

A fulminant edema quite different from the one just described sometimes occurs in influenza, killing its victims in from twelve to twenty-four hours. The patients—those I have seen or know about were girls and young women—have a grayish, cyanotic, or slaty color, are profoundly toxic, stuporous, or comatose. The rectal temperature is high, the circulation in collapse, the extremities cold; the lungs show fine râles, but not always in great abundance. There may be frothy salmon-colored expectoration; not rarely death occurs before anything is brought up. Consolidation of the lungs cannot be demonstrated; the virus causes death before pneumonia has time to develop.



8. There is another form of edema analogous in its manifestation, which I would call **postaspirational**. I can best illustrate it by describing to you an experience I had when I was a beginner in medicine. I tapped a woman who as the result of a mediastinal tumor had had a one-sided hydrothorax for a long period of time. As I was cleaning up the apparatus in an adjoining room, preparatory to leaving the house, I was suddenly recalled to the bedside of the patient. I found her in a terrifying condition. Her face had an agonized expression; her color was purple; she was sitting up gasping for breath; the death rattle seemed to be in her throat. She was practically pulseless at the wrist; was covered with a cold sweat—not so cold as mine, however; I thought she was dying, and with her one-third of my practice. While I was watching her she suddenly spat up a huge quantity of syrupy looking fluid, exactly like that in the aspirator bottle—I had gotten about a quart or more of fluid. My immediate conclusion was that I had punctured the lung. I did what I could for the patient, and either because of what I did or because—as Jerome Cardan might have said, “my familiar spirit intervened”—she got well of her attack. I began to wonder what had happened, never having seen or heard of such a condition before. I boiled the expectorated fluid and that which I had drawn off with the aspirator. They both coagulated solidly; they seemed to be the same fluid, confirming me in my first impression that the lung had been punctured. But when I began to think more calmly and looked up the subject—and I may say that I had a hard time finding anything about it—I found that similar cases had been reported; one by the late Dr. William Pepper. Dr. Pepper’s patient died. After due consideration I came to the conclusion that I had not punctured the lung, but that the thing I had seen was an acute edema of the lung. The lung expanding in a vacuum after long compression became suddenly intensely congested and overfilled with blood. Because of the long compression the vessels in the lung were altered and abnormally permeable; the fluid therefore leaked out, and being blood-serum, just like that in the pleura, it was albuminous and



coagulable; hence the term "albuminous expectoration" under which the case was reported. To distinguish the edema in such cases and to indicate its nature I have called it **edema by recoil**. Fortunately, it is as rare as it is terrifying. Why it happens in the exceptional case is difficult to say. It is more likely to occur in cases in which the pleural effusion with compression of the lung has existed for a long time; also if there is some cardiac embarrassment, such as myocardial weakness or pericarditis. When tapping<sup>1</sup> in such cases one should always be prepared for it. Otherwise, the patient is likely to die. I will discuss the measures later on. Similar edema has been observed in rare instances after tapping of the abdomen. The edema is probably due to the same cause, the lung having been crowded and compressed for a long time by a large peritoneal effusion.

An edema that is rather unimportant from a human standpoint perhaps, but which throws some light on edema in general, is that due to adrenalin. If you are familiar with the experiments of Meltzer, you may know that adrenalin in animals may produce an intense edema that may be fatal. Whether adrenalin has a similar effect in man is a mooted question, which was rather vehemently agitated in a discussion on pulmonary edema at one of the meetings of the American Association of Physicians (Transactions, 1906).

There is another form of edema that I would speak of under this head, and that is **angioneurotic edema**. While this edema is most common in the skin, there is no reason why it may not occur anywhere where there are blood-vessels. There is no anatomic difference between edema of the lungs and edema of the skin; in the skin the serum following the path of least resistance oozes into the interstices; the same thing happens in the lungs, where it passes into the vesicles or into the interstitial tissue outside of the capillaries.

An acute edema sometimes follows suddenly after *operation*. I do not mean the not infrequent postoperative crackling at

<sup>1</sup>In a number of the American Journal of the Medical Sciences soon to appear I am reporting a recent case of acute pulmonary edema following tapping.

the bases, but a real apoplectic edema. It may be angioneurotic in origin.

**Treatment.**—The treatment of the passive form of edema of the lungs is not important. We do not treat the edema directly, but the underlying condition—the failure of the circulation. Sometimes, as I have said, the edema may be lessened or relieved by setting the patient upright with a bed-rest or by getting him out of bed.

The acute toxic edema so commonly found in influenza and in pneumonia is sometimes very hard to deal with, as those of us who passed through the recent influenza epidemic remember only too well. The remedy that seems to do most good is atropin in large doses— $\frac{1}{75}$  or  $\frac{1}{80}$  grain. Sometimes bleeding is helpful, but we have often bled in the Philadelphia General Hospital without results. Cardiac stimulation is also of value. I did not see much benefit from glucose solution, but as we usually have given it by bowel and not intravenously, as has latterly been recommended, perhaps the disappointing results are to be ascribed to a faulty method.

In the treatment of the apoplectic form and that following aspiration of the chest, the best remedial agent is a hypodermic injection of morphin and atropin. The effect is sometimes magical and is in part due to the fact that morphin relieves the intense psychic unrest and fear. The second thing of value is dry cupping—a half-dozen cups to the front and to the back. This is often productive of instantaneous relief and I have great faith in it. Cupping is a very simple procedure and no complicated apparatus is necessary. A number of applicators are made out of match sticks and the inside of a small whisky or wine-glass is moistened with an applicator dipped in alcohol. The alcohol is then lighted, and before the flame blazes down the glass is applied evenly to the chest. The cups are left on until the skin has a purple hue.

Another excellent measure, useful especially in hypertension cases, is bleeding. I have employed this several times, particularly in cases in which cerebral symptoms were an accompaniment.

In the apoplectic cases it is not only necessary to treat the attacks but also to find measures for preventing their recurrence. That can only be done by treating the underlying condition, and as this in the majority of cases is hypertension or nephritis and hypertension, treatment for these conditions must be instituted. It is, however, not my purpose to go into the details of this subject, nor can I discuss the treatment of mitral stenosis and myocarditis, which are also capable of producing the edema.

To guard against postaspirational edema it is in the first place necessary in all cases where it is a possibility, as in long-standing large effusions with myocarditis or pericarditis, to draw the fluid off slowly and with the least possible shock to the patient. Secondly, one should always remain near the patient's bedside for at least half an hour after the tapping. Thirdly, on the first sign of edema the patient should receive a hypodermic injection of morphin and atropin. Dry cups should then be applied and digitalis given. If oxygen is at hand it may be used, but when the lung is so full of fluid and breathing so embarrassed, it is hard for the patient to get any benefit from oxygen.

I hope that I have in a small measure awakened your interest in the subject of edema of the lungs. It is one that will well repay clinical and experimental study.

## CLINIC OF DR. H. R. M. LANDIS

UNIVERSITY HOSPITAL

---

### MENINGITIS

**Meningism; Meningitis of Undetermined Origin; Tuberculous Meningitis in Adults; Meningococcus Meningitis; Pneumococcus Meningitis; Syphilitic Meningitis; Typhoid Meningitis; Illustrative Cases.**

THE presence of meningeal irritation is in the great majority of instances a simple matter to determine, as both the symptoms and signs are easily recognized. The cause of the irritation, however, is not always so easily recognized. Signs of meningitis plus certain changes in the spinal fluid do not necessarily mean that true meningitis is present. In a recent study of the spinal fluid in cases not resulting in meningitis Herrick and Dannenberg (Jour. Amer. Med. Assoc., Nov., 1919) found that the spinal fluid was often under increased pressure, contained more cells than are ordinarily accepted as being normal, and, in addition, showed an increased amount of globulin. In most of the cases, but by no means all, there were present also the signs of meningismus or meningism. Among the diseases studied by Herrick and Dannenberg were lobar and bronchopneumonia, influenza, tonsillitis, the exanthems, sepsis, pleurisy, etc. The important point brought out in their paper is that too much reliance on the spinal fluid findings has hitherto been placed on data obtained from definitely inflamed meninges. More information is needed on the character of the spinal fluid in normal individuals and in those suffering from various diseases. A diagnosis of true meningitis must rest partly on clinical evidence and partly on the laboratory findings.

**Meningism (Meningismus, Meningitis Serosa Dupre).**—In many instances the clinical picture of meningism is not unlike that which occurs in true meningitis. Thus there may be headache, some stiffness of the neck, eye signs, etc. As a rule, examination of the spinal fluid will distinguish these cases of pseudomeningitis or meningism from true meningitis. Although the amount of the fluid and the pressure are often increased in these cases, the color is unchanged, the number of cells is normal or only slightly increased, and, most important, the bacteriologic tests are negative.

In meningism the symptoms may disappear in a day or so, but occasionally the evidences of cerebral irritation may last for a week or more. Recovery is the rule.

If the symptoms are unduly prolonged one should be alert to the fact that they may be due to tuberculosis, syphilis, or poliomyelitis, as these conditions may, in the beginning, show nothing more than an increase in the amount of and an increase in the pressure of the spinal fluid.

**Meningitis of Undetermined Origin.**—From time to time cases are admitted to the wards in which the evidence of meningitis is clear, but in which the cause cannot be clearly established. The following cases are examples of meningitis of obscure origin:

**CASE I.**—A male, aged twenty-two years, was struck on the head with a bottle during a drunken brawl. Two days before his admission to the hospital and four days after the injury he developed a persistent headache. He was stuporous on admission and lay curled up on one side. The site of his injury was located by a small edematous area on the scalp just to the right of the median line and anterior to the coronal suture. The neck was stiff and the head a little retracted. The reflexes were noted as being active. Examination of the eyes showed the pupils to be contracted, the vessels on the edge of the disks injected, and the edges of the disks hazy. At a later examination the change in the disks were even more marked.

There was no fever. The pulse-rate was slow—at times

falling to 48 per minute. The leukocyte count was 8900. The blood-pressure showed nothing abnormal. At lumbar puncture clear fluid was removed under pressure. At the first tapping the cells were not increased, but at a later one numbered 314. Examination of the fluid failed to reveal any bacteria and the globulin and Wassermann tests were negative. Two weeks after admission the evidences of cerebral irritation had entirely disappeared, except for some haziness of the nerve heads, and the patient was discharged. The final diagnosis in this case seemed to point to a serous meningitis—the result of the trauma.

In this connection mention might be made of another case in which trauma was a factor. The patient was struck over the left temple by a strong air blast. The following day he became weak and developed a sharp shooting pain in the left temple; later this pain became generalized over the entire head. Shortly after the pain developed he had a chill, and this was repeated nearly every other day for two weeks. During this time he had a continuous fever. For the first two weeks of his stay in the hospital the clinical picture was very confusing. The feeling, however, was that the patient was suffering from some cord lesion. Finally, the symptoms became more and more suggestive of meningitis. A lumbar puncture was done and the laboratory findings showed the case to be one of meningococcic meningitis. The patient recovered. It is not clear in this case whether the trauma produced a point of lowered resistance in the meninges, or whether the primary symptoms were simply due to the trauma and those which developed later were the result of an independent meningococcic infection.

CASE II.—A male, aged thirty-two years, sought relief from a headache which he said had been almost continuous for six months and had been preceded by an attack of malaria in the tropics.

Examination showed doubtful signs at the right apex, but no respiratory symptoms. The reflexes were normal. There was some stiffness of the neck. The blood-pressure was 110-68 and the leukocyte count 6500. The pupils were unequal and

the right disk a little paler than normal. Two lumbar punctures were made; each time cloudy, straw-colored fluid was removed. The first cell count was 622, with 98 per cent. lymphocytes, and the second 859, with 93 per cent. lymphocytes. The Wassermann test was negative. The spinal fluid was negative to all tests, including animal inoculations. Two weeks after his admission the patient left the hospital free of all symptoms.

The long duration of the headache, the suspicious right apex, and the lymphocytosis pointed strongly to a tuberculous origin of the trouble, but this failed of proof even after guinea-pig inoculation.

CASE III.—A man aged sixty years, with an antecedent history of a number of serious illnesses, was admitted to the hospital in a slightly stuporous condition. It was stated that six days previously he had first noticed some weakness of the left side. Shortly after admission it was noticed that the lower part of the left face was paralyzed. The reflexes on the affected side were increased. Examination of the eyes showed some ptosis of the left eyelid. The margin of the right disk was hazy and the nerve head hyperemic.

At lumbar puncture clear fluid was obtained containing from 5 to 6 cells per cubic millimeter. The globulin and Wassermann tests were negative, as were also the bacteriologic tests.

Because there was a history of a chronic ear discharge a brain abscess was suspected, and the patient was referred to the surgical service. The right motor cortex and the major portion of the right temporal lobe were exposed. There was no evidence of increased tension. A number of subdural adhesions were present; these were noted anterior and posterior to the opening as well as toward the base of the brain. A thorough exploration in every direction failed to show either an abscess or a tumor. The surgeon's belief was that the symptoms were due to a meningitis. Fluid obtained from the brain was sterile.

The patient died a month later without any additional light on the cause of the trouble.

## TUBERCULOUS MENINGITIS IN ADULTS

It is well known that tuberculous meningitis is a very common cause of death in infants and young children. Thus, of 8877 deaths from tuberculosis of all kinds in children under five years of age, 3347 were due to meningitis (Cobbett). It is not so well recognized, however, that tuberculous meningitis is not infrequently the terminal event in adults suffering from some form of tuberculosis, particularly pulmonary tuberculosis.

During a period of two or three years there were seen at the University Hospital and the White Haven Sanatorium 23 cases of tuberculous meningitis in adults. The primary focus in these cases was as follows: lungs in 19; kidney in 2; caries of the vertebrae in 1; and in one the location was not determined.

Of these 23 cases, 14 were males and 9 females; 20 were white and 3 colored; the ages ranged from fifteen to fifty-two years.

**Onset.**—In 10 cases the onset was acute; in 9 it was gradual, and in 4 it was protracted. Those with a protracted onset are worthy of special comment. The histories of the cases in this group seemed to indicate quite clearly that the evidences of meningeal irritation had been present persistently or intermittently for two and a half, three, six, and nine months respectively. For example: A young woman aged twenty-two had had one brother die of tuberculous meningitis. Nine months prior to her admission to the hospital she began to suffer from severe attacks of headache which would come on gradually and last for twenty-four hours. These attacks, for the first three months, would occur about twice a month. They tended, however, to become more severe and more frequent. Three months from the onset of these attacks she was severely exposed to cold and wet weather, and following this the headache became continuous, the neck muscles became stiff and painful, and the head markedly retracted. In addition, she was delirious and partly unconscious for a week; there were no paralyses.

The pain and rigidity in the neck disappeared, but the headache persisted and was almost continuous. In addition to other evidences of meningitis on admission she had pain on pressure over the lumbosacral region and several of the thoracic vertebrae.



Although cases with a protracted onset are atypical, it is well to remember that they do occur, and that in cases of pulmonary tuberculosis the premonitory headache is not to be ascribed always to eye strain or sinus trouble.

**Duration.**—As a rule the length of time between the appearance of the initial symptom and the termination of the disease is between ten and thirty days; the average time being about three weeks. As already pointed out, however, cases are occasionally seen in which months elapse between the appearance of the initial symptom and death.

**Initial Symptoms.**—*Headache* is the most constant of the early symptoms. It was the first manifestation in 18 cases and was present in all sooner or later. There is nothing characteristic of the location of the headache; it may occur generally all over the head, or it may be located in the frontal or occipital regions. It is always severe, persists in spite of the use of one of the coal-tar products, and usually requires an opiate for its relief.

The disease may be ushered in with *delirium*; this occurred in 7 cases, and in all it was noted at some time during the course of the disease. In a few instances the initial manifestation is a more or less marked *stupor*. In 16 cases stupor was a prominent symptom during the course of the disease. *Insomnia* alternating with stupor was noted in 3 cases.

In 11 cases *nausea* and *vomiting* were among the initial symptoms. It is usually taught that vomiting, due to a cerebral lesion, is projectile in type, but in this group the vomiting in 10 was of the ordinary gastric type and in but 1 was it cerebral.

*Rigidity of the neck muscles* with or without retraction of the head is a characteristic sign of meningitis; in addition, the muscles may be painful on movement. Rigidity was noted early in the course of the disease in 11 cases.

**Progress of the Disease.**—One or more of the symptoms above referred to may be the first evidence of the trouble, but as the disease progresses they are nearly all present at one time or another. In addition, other signs appear.

**Eye Signs.**—The most important of these are signs referable

to the eyes. Paralyzes of the ocular muscles are extremely common in tuberculous meningitis in contrast with the epidemic variety of the disease in which ocular paralyzes are relatively infrequent. The frequency of these changes in the tuberculous form of the disease is to be attributed to the location of the lesion at the base of the brain. Among the eye changes noted in this series were paralyzes of one or more of the ocular muscles in 15 cases; irregular pupils in 7 cases; nystagmus in 4 cases; optic neuritis in 3 cases; ptosis of the eyelids in 3 cases; injection of the conjunctiva in 5 cases; photophobia was noted in but one instance. The presence of choroidal tubercles is cited in the text-books as a valuable diagnostic sign, but it is only rarely that they are found. The eye-grounds were examined in only 4 of these cases and in none of them were tubercles seen.

**Paralysis.**—As I have already pointed out, paralysis of one of the ocular muscles is a frequent occurrence in tuberculous meningitis and relatively infrequent in the meningococcic variety. The reverse is true in regard to the paralysis of the muscles of the extremities. In meningococcic meningitis contractions and paralyzes are frequently seen in cerebrospinal meningitis during the course of the disease, and these may be permanent. In the tuberculous variety they are relatively infrequent. In one case there was a partial loss of power of the muscles of the left upper and lower extremities; in one there was marked flaccidity of the muscles of all the extremities, and in a third there were convulsive twitchings of the muscles on one side of the body. *Abdominal rigidity* was noted in these cases. The *reflexes* may be abolished when the case is first seen, or they may be exaggerated at first and later disappear.

The histories indicate that Kernig's sign was looked for in 17 of the 23 cases; it was present, usually to a marked degree, in 14 and definitely absent in 3. Babinski's reflex is occasionally present and the Brudzinski reflex more often so.

**Hyperesthesia** was present in 3 cases.

**Constitutional Symptoms.**—Occasionally there is little or no fever, but this is rare. There is nothing characteristic about the fever curve. As in the majority of the cases, the meningitis is

a terminal event in either chronic or acute pulmonary tuberculosis, the fever present is usually of the type seen in these conditions. It may, however, be increased or diminished with the onset of the meningitis.

There is a very prevalent belief that marked slowing of the pulse or *bradycardia* is a characteristic feature of meningitis. As a matter of fact, marked slowing of the pulse is unusual, and even a relative-fall in the pulse-rate is not common. Of the 23 cases under discussion, a low pulse-rate was noted but once; in 6 cases it was slightly reduced from the rate previously existing; in the remainder it remained high or was markedly accelerated.

*Cheyne-Stokes* type of breathing was noted in 3 cases. *Biot's* breathing, a type characterized by a series of rapid but equally deep respiratory movements followed by sudden apnea, was not noted. As few are familiar with this alteration in the respiratory function, it may have occurred and not have been noted. When present it is said to be almost pathognomonic of meningitis.

The *urine* is usually of the febrile variety. A trace of albumin is commonly present; casts may also be found.

The blood-pressure readings were normal in all the cases examined, with one exception; in this case there was a systolic pressure of 215 and a diastolic of 140.

**Laboratory Examinations.**—The *leukocyte count* varies greatly; it may be normal or only slightly increased. When present the leukocytosis is not usually very high. Of 13 cases with a record of a white cell count, 4 were normal, 4 were moderately increased (12,000), and in 5 the count ranged from 17,000 to 20,000.

**Spinal Fluid.**—While in most cases of tuberculous meningitis the diagnosis can be made with reasonable certainty because of the easy detection of the primary focus, the only way we can be certain of the etiologic factor is by examining the spinal fluid. This, of course, applies with even greater force to other forms of meningitis.

The spinal fluid in tuberculous meningitis is usually under increased pressure, although an initial high pressure may

diminish during the terminal stage. The fluid is usually clear, shows a white foam on shaking, and, after standing, a pellicle forms, the pellicle being suspended in the center of the tube. The cell count, in contrast to the acute infectious forms of meningitis, is not very high. The lowest count in this series was from 17 to 30 in one case, and the highest 620. The cells may be entirely lymphocytes or predominantly so. Tubercle bacilli are easily demonstrated in the spinal fluid in some cases; in others only after patient search, and in many instances only after guinea-pig inoculation. The tubercle bacilli are, as a rule, most easily found in the pellicle. While in the majority of cases of tuberculous meningitis the above description holds true, there are certain variations met with. The fluid may be turbid, this being the case in 4 of the 23 cases. Occasionally, instead of there being a lymphocytosis, the cells may be entirely polymorphonuclear in type, especially in the early stages of the disease. In a case seen by Dr. O. H. P. Pepper, and proved at autopsy to be tuberculous meningitis, the spinal fluid was turbid and the cells, numbering 600 per cubic millimeter, were practically all polymorphonuclears.

In addition to the above methods, which are simple and easily carried out, the fluid in obscure cases may be subjected to tests for globulin (preferably by the Noguchi method), the permanganate index, and the Lange gold chlorid test.

**Termination.**—Although an occasional recovery is reported from tuberculous meningitis, the validity of the diagnosis is usually open to question. The disease may be said to be invariably fatal. Of the 23 cases under discussion, all died.

**Summary.**—Tuberculous meningitis, as seen in adults, is most commonly associated with tuberculosis of the lungs. The first symptom to appear is usually headache. Following this there is delirium, sometimes accompanied by vomiting. Within a few hours these symptoms may disappear with the exception of the headache, which persists. The reflexes are usually normal at this time. The headache may be replaced by stupor, which later alternates with delirium. The next symptoms to appear are usually ocular (irregular pupils, diplopia, etc.). Later the

muscles become weakened, the reflexes disturbed, and delirium or coma becomes marked.

From the laboratory side the most important diagnostic points are: Increase in the pressure of the spinal fluid; clear fluid; increase in the number of cells with a relative or complete lymphocytosis; the presence of tubercle bacilli in the fluid, or positive inoculation tests; and an increase in the amount of globulin.

#### MENINGOCOCCUS MENINGITIS

Of the cases in this group, numbering 19, all were of the sporadic type; 13 were males and 6 females; 18 were white and 1 colored. The ages ranged from seventeen to sixty-one years.

**Onset.**—The onset in this form of meningitis is usually abrupt and indicative of an acute infectious process. A chill or chilly sensations occurred in 8 of the 19 cases; headache in 17; vomiting in 11; and rigidity of the neck, as an initial symptom, in 2. Occasionally a fulminant case is seen in which an apparently healthy individual falls unconscious, as though from an apoplexy, and death occurs within a few hours. On the other hand, the onset may be gradual and the course of the disease more or less prolonged.

**Symptoms.**—As the disease progresses the headache tends to increase in severity or is replaced by alternating delirium and stupor. In 16 of the cases delirium was present, and in 8 stupor. Rigidity of the neck was noted in 12 and, in addition, the neck muscles were very painful in 3. Convulsions occurred in 3 cases. The legs were flexed in 5 cases. In 1 case there was unilateral paralysis of the facial muscles. In but 1 of the 12 cases ending in recovery was there a permanent paralysis (leg muscles). Kernig's sign was well marked in 14; in 7 the reflexes were noted as being normal and in 2 they were exaggerated.

**Eye Changes.**—As already pointed out, the eye signs are not nearly so prominent a feature in meningococcus meningitis as they are in the tuberculous type of the disease, especially in regard to the paralysis of the eye muscles. Only 2 of the 19 cases showed paralysis of one of the ocular muscles, and in one nystagmus was noted. In 3 examination of the eye-grounds showed

a choked disk. Photophobia was complained of in 5, and in 3 pain in the eyeballs was present.

**Skin.**—Certainly so far as the sporadic cases of meningococcus meningitis are concerned a skin rash is not a prominent feature. In 6 of the 19 cases rashes of various sorts were noted; in 8 the tache cérébrale was marked; and in 5 there were labial herpes.

**Fever.**—The fever curve is not characteristic, but is subject to numerous variations. Occasionally there is no fever, as noted in one case in this series. In one case the temperature rose to 108° F.

Marked slowing of the **pulse** was noted in but one of the cases; a relative bradycardia was present in 3; in the remainder the pulse-rate was rapid and proportionate to the degree of fever present.

In 13 of the 19 cases both **albumin** and **casts** were present. The **blood-pressure** estimations varied greatly, ranging from 109 to 180 systolic and from 50 to 100 diastolic.

**Laboratory Examinations.**—A well-marked *leukocytosis* is present in practically all cases of meningococcic meningitis. In 3 cases the initial count was 8800, 9600, and 11,200; later the counts in these cases were 24,000, 15,800, and 30,000 respectively.

**Spinal Fluid.**—The spinal fluid is practically always increased in amount, and this is true throughout the course of the disease. If a diminished amount is present it is usually the result of an obstruction somewhere in the spinal canal. The fluid also flows under pressure. In nearly all of the cases the fluid is turbid and remains so throughout the disease. It occasionally happens, however, that the fluid is clear and colorless in the early stages of the disease. In 2 of the 19 cases the fluid was clear; in 17 the fluid was turbid, and, in addition, in 4 of the cases it was bloody. Later in the course of the disease the fluid becomes yellowish green or even green as the result of the presence of meningococci.

On standing, a sediment usually forms. The **cell count** is invariably high. In the cases under discussion it ranged from 300 to 10,240 per cubic millimeter. The predominant type of cell was the polymorphonuclear leukocyte, the percentage ranging from 60 to 94. Lymphocytes are also present.

The meningococci can, as a rule, be readily demonstrated in smears made from the spinal fluid. They are usually seen within the leukocytes, but may be found also outside the cells. Early in the disease the organisms may be scanty and difficult to detect, but after one or two doses of serum they are apt to appear in large numbers.

Meningococci were demonstrated in smears from the spinal fluid in 15 of the 19 cases; in another case the organisms were recovered from the blood.

In addition, the tests for globulin are strongly positive. Sugar is either absent or greatly diminished in amount.

**Results.**—Twelve of the 19 cases made a perfect recovery; in another case ending in recovery the leg muscles were paralyzed. The remainder (7) died—a mortality of 36.9 per cent.

#### PNEUMOCOCCUS MENINGITIS

Tuberculous meningitis and meningococcic meningitis are by far the most common types of the disease even when the latter does not occur as an epidemic. Although the tubercle bacillus and the meningococcus are the most frequent etiologic factors, meningitis due to other causes is seen relatively frequently. Perhaps the next most common form is that due to the pneumococcus.

Two examples of this type of meningitis have been seen in the University Hospital within the past year or so.

**CASE I.**—The patient was admitted complaining of headache and slight soreness and rigidity of the neck.

He stated that the illness had started one week previously with a cold and pain in the chest. This was followed shortly by headache and some pain and rigidity of the neck muscles. On admission to the hospital the patient was stuporous, restless, and discontented. The pupils were dilated, the sclera was injected, photophobia was present, and, in addition, there was slight horizontal nystagmus. The reflexes were exaggerated. Kernig's sign was well marked, but Babinski's sign was absent. Herpes was not present. Examination of the lungs was negative.



A lumbar puncture was done and 15 c.c. of slightly cloudy fluid were removed drop by drop. There were 3520 cells per cubic millimeter. The cells were practically all polymorphonuclear in type, and, in addition, there were many diplococci in and between the cells. Examination of the blood showed a leukocytosis of 18,200 with 88 per cent. neutrophils. At the second lumbar puncture the following day the fluid spurted out and was much cloudier than at the first tapping. There were very few leukocytes present, but a pure culture of capsulated diplococci was obtained, and these were identified in the laboratory as belonging to the Type I group.

A third puncture was done and bloody fluid removed drop by drop. The patient gradually grew worse, and some hours before death became very cyanotic, with labored and stertorous breathing.

CASE II.—A colored man aged thirty-eight was admitted to the hospital in an unconscious condition. The history given by friends was that three weeks before he first developed some pain in the lower left chest, but kept on working for eleven days. He finally had to stop work because of the pain and attacks of dizziness. Four days before admission his neck became stiff, and a few hours later he developed headache. He became unconscious the night before entering the hospital. When first seen it was noted that about every five minutes there were attacks of trembling and jerking movements in all of the extremities, but most marked in the arms. The reflexes were all exaggerated and the muscles were thrown in a tremor by tapping or the least irritation.

The blood-pressure showed a systolic reading of 215. Examination of the blood showed a leukocytosis of 35,200, 82 per cent. being polymorphonuclears.

The temperature ranged from 103° to 105° F.; the pulse-rate from 110 to 170; and the respiratory rate about 60.

Examination of the lungs showed some exaggeration of the expiratory sound and an increase in voice sounds at the left base.

A lumbar puncture was done and 8 c.c. of turbid yellow fluid removed. The cell count was 2020 per cubic millimeter, all



being polymorphonuclears. A stained specimen showed Gram-positive extracellular organisms, joined end to end, oval in shape, and surrounded by what seemed to be a capsule. The laboratory reported that there were no tubercle bacilli or meningococci, but a pure culture of Gram-positive diplococci.

Levinson states that when the pneumococci are joined end to end they may be confused with streptococci. When the former are present the pellicle contains more fibrin and the capsule can usually be made out. The final diagnosis in such cases, however, must rest on the culture and the agglutinative properties of the organism. It is to be borne in mind that both organisms may be present. The globulin tests are strongly present in both streptococcus and pneumococcus meningitis.

#### SYPHILITIC MENINGITIS

Irritation of the meninges is quite a common manifestation in lues, but in most instances it is associated with involvement of the central nervous system as well. As a rule, it is a chronic condition and quite commonly escapes detection. Occasionally an adult case is encountered in which the clinical picture is almost identically that seen in meningitis due to other causes. It is most apt to be confused with tuberculous meningitis or poliomyelitis. The following case is an excellent example:

A male, age thirty-three, was admitted to the hospital because of pain in the back of his head and neck. His trouble began four weeks before with a dull headache in the lower occipital region. The headache at first was not constant, but gradually became worse. Two weeks before his admission the headache became so severe he had to stop work. The headache was always occipital and much worse at night. At this time he had to make an effort to "collect his thoughts," felt "heavy," and sat about the house without energy enough to do anything. Coincidentally with the appearance of the headache his eyes, especially the left, felt tender and sore.

Although he denied any venereal infection, it was learned later, from the Venereal Dispensary, that he had been seen there six months before with a primary lesion. He had been given

several doses of arsphenamine, but was irregular in his attendance and was drinking heavily.

Examination of the heart, lungs, and abdomen showed nothing abnormal. The reflexes were normal; Kernig's sign was slightly present. Examination of the eyes showed the pupils moderately contracted, but equal, and injection of the conjunctiva. There was no ocular paralyses or other abnormalities.

Two weeks after his admission he became drowsy and irrational. Later he became delirious and had to be restrained. At this time examination of the eye-grounds showed the disks to be slightly hyperemic, with some blurring of the margins, although still well defined. The retinal veins were full, but not tortuous. The blood Wassermann was four plus. The leukocyte count was 6200.

A lumbar puncture was done and 25 c.c. of clear fluid removed under great pressure. The cell count was 21 per cubic millimeter, 98 per cent. being lymphocytes. The Wassermann reaction was strongly positive. Tests for globulin were positive. There were no tubercle bacilli present and nothing was obtained from cultures. In such cases the fluid should also be examined for the presence of spirochetes.

The patient cleared up rapidly under the use of salvarsan and was discharged six weeks later apparently well. He was admitted six weeks after his discharge because of an acute infection, and at this time showed no meningeal symptoms.

#### TYPHOID MENINGITIS

This is a rare condition. A case, meeting all of the bacteriologic requirements, was seen in the University Hospital some years ago and reported by Lavenson (University of Penna. Med. Bul., April, 1908). Following is an abstract of this report:

A female, age twenty-six, was admitted to the University Hospital suffering with severe frontal headache, vomiting, photophobia, and fever. For several weeks prior to the onset of her illness she had been nursing her brother through a severe attack of typhoid fever. Four days before her admission to the hospital she was suddenly seized with an agonizing frontal headache,

associated with vomiting, photophobia, and great muscular weakness. There was also suppression of the urine.

On admission her temperature was 103° F. During the last eight days of life it ranged from 99.3° to 103.3° F. The pulse-rate was 80. She was slightly stuporous, but not delirious, and was evidently suffering extreme pain. The pain was increased by pressure over the forehead. There was no rigidity of the neck and no palsies. The knee-jerks were normal and Kernig's sign could not be elicited. There was no eruption or enlargement of the spleen. The Widal reaction was marked and several leukocyte counts averaged 20,000 cells. A lumbar puncture was made and cultures made from the fluid showed the typhoid bacillus. She died eight days after admission.

The autopsy showed no evidence of typhoid fever in the intestines or spleen. Over the convexity of the right cerebral hemisphere, slightly anterior to the premarginal fissure, there was a collection of pus in the pia-arachnoid. Slightly below and anterior to this area was a smaller collection. The frontal portion of the left convexity showed a small focus of the same nature.

The ventricles contained a moderate amount of light yellow, slightly turbid fluid. There was no evidence of tubercles anywhere. Smears from the pus showed a large number of Gram-negative bacilli having the morphologic characteristics of typhoid bacilli and a few larger Gram-positive bacilli.

The infection of the meninges in this case may have occurred through the nose, the organisms being conveyed by the fingers.

## CLINIC OF DR. GEORGE WILLIAM NORRIS

PENNSYLVANIA HOSPITAL

### SYPHILITIC AORTITIS

**Presentation of Two Cases: Illustrating the Effects of Syphilis Upon the Aorta. One Showing the Development of a Large Aneurysm. Full Discussion of Treatment and Management.**

THE first patient I have to present to you today is a laborer, forty-two years old, who was admitted to this hospital complaining of "weakness in the stomach," dyspnea on exertion, and edema of the legs. The symptoms of the present attack began two weeks ago.

*Past History.*—He has had the usual diseases of childhood, no accidents or operations. No rheumatic fever. At the age of seventeen he had a chancre, but never noticed any secondary symptoms. He never had gonorrhea. He drank heavily (whisky) between the ages of twenty and forty years.

*Family History.*—His father, grandfather, and an uncle died of Bright's disease in the sixth decade.

*Present Illness.*—He always enjoyed good health until April, 1918, when he first noticed a sensation of epigastric "goneness" and dyspnea on exertion. He was twice admitted and discharged from hospitals for these symptoms. In October, 1918 he developed, in addition, swelling of the legs. Twice since then he has been in hospitals, exclusive of his present admission.

You see before you a pale but well-developed and nourished white man, propped up in bed, his head nodding with each heart-beat. He is markedly edematous. His lips are pale, the teeth poorly kept, several being carious. You will note marked arterial pulsation in the vessels of his neck and distended pulsating jugular veins. There is no adenopathy or tracheal tug. The

chest is somewhat emphysematous, shows marked inspiratory retraction of the lower intercostal space—a valuable sign of deficient pulmonary aëration. On the blackboard you will see a diagrammatic representation of his physical signs (Figs. 238, 239).

You will note that the heart is greatly enlarged, especially to the left. This border is, however, outlined with uncertainty,

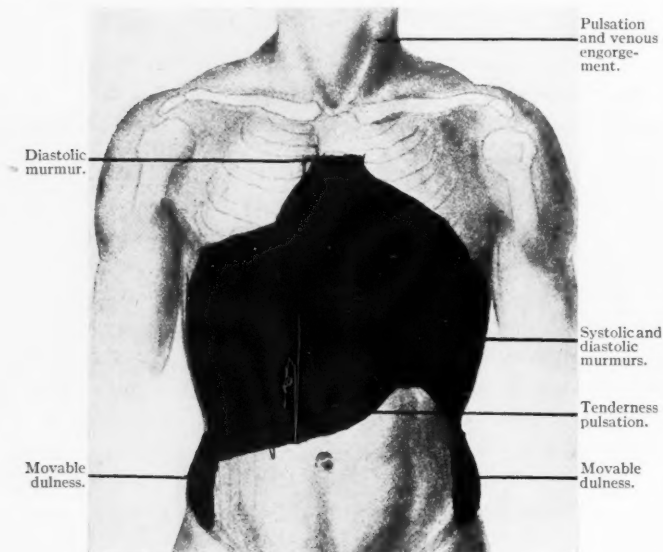


Fig. 238.—Pulse 100; blood-pressure  $\frac{175}{95}$ ; radial pulses equal, synchronous, collapsing. Arteries thickened. Occasional extrasystoles. Breath sounds exaggerated above dull areas; absent beneath the same.

owing to the presence of the left-sided hydrothorax. The diastolic murmur at the base is evidently a result of aortic insufficiency. No other condition except shock causes a diastolic pressure of 35 mm. This murmur is also heard over the lower precordium, but, in addition, there is also a systolic murmur. The latter is assumed to be the result of mitral insufficiency, since it is not heard at the base or in the vessels of the neck. It may,

however, be in part due to tricuspid insufficiency, a lesion which we can confidently diagnosticate by the presence of congested lungs, hydrothorax, a pulsating liver, ascites, and edema of the legs.

Posteriorly you will see that the hydrothorax, which is evidenced by the dulness, increased tactile resistance, and absent breath sounds, is greater on the right than on the left side. This is commonly the case when a hydrothorax results from cardiac dilatation, and is apparently due to the greater compression

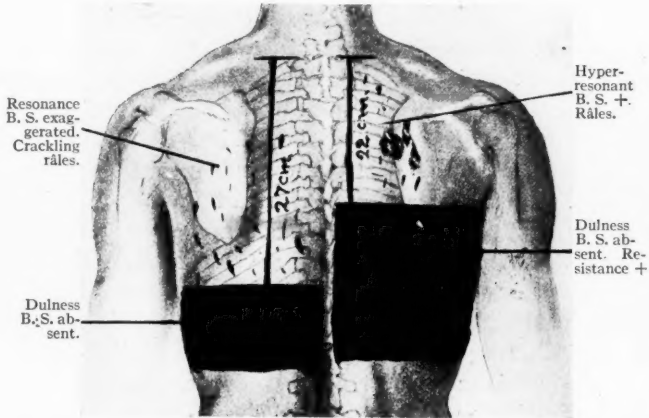


Fig. 239.—Physical signs indicating a large right- and a small left-sided pleural effusion. Congestion of the lungs (râles).

which is exerted by the right auricle upon the pulmonary veins on that side. On the left side, in order to produce an equal degree of pressure upon both the upper and lower veins, one must have a dilatation of the left auricular appendix and of the left ventricle.

The sequence of events in this case has been, I think, as follows:

Syphilitic infection twenty years ago producing an aortitis which developed insidiously, and unchecked by treatment. Gradually, as the process spread downward, the aortic leaflets

became involved, producing an insufficiency and cardiac hypertrophy. Then, as a result of age, a laboring occupation, and continued syphilitic disease, the myocardium became weakened. The mitral sphincter relaxed and extra work was thrown upon the right heart. At this time the patient suffered only from dyspnea on exertion. Finally, the right heart dilated under the strain, a relative tricuspid insufficiency became established, and the characteristic symptoms of broken compensation set in.

DR. NORRIS: Now what would you suggest in treatment?

A STUDENT: Digitalis.

DR. NORRIS: Yes, but perhaps of even greater importance, rest in bed, with a light diet, restricted in fluid. In some cases limiting the total intake per day to 1 pint of milk produces excellent results. What about salt restriction and diuretics?

A STUDENT: What is the renal condition?

DR. NORRIS: Quite right. It depends on the state of the kidneys. If the edema is due or in part due to nephritis, we would restrict the sodium chlorid intake and perhaps administer diuretics for short and usually intermittent periods of time, but when the urinary output is diminished as a result of passive congestion, we rely upon digitalis or allied drugs to improve the circulation through the kidneys and thus increase diuresis.

Now in this case the urinary examination shows a specific gravity ranging between 1016 and 1018, a trace of albumin, and an occasional hyaline cast.

The phthalein excretion was on admission only 30 per cent. in the first two hours it is true, but this index does not help us to differentiate between congestion and nephritis, as it is reduced in each instance. Further, you will note from the graphic chart of fluid intake and urinary output that the latter not only exceeds the former, but is increasing progressively under digitalis. When first admitted the patient was only voiding 20 ounces daily. The quantity has now increased to 60.

I believe that we are dealing with passive renal congestion. The examination of the blood shows nothing of importance except a positive Wassermann reaction. The x-ray shows no evidence of thoracic aneurysm.



The next patient I have to show you is a negro laborer, thirty-nine years of age, who comes to us complaining of chest pain. This symptom, which was first noticed but six weeks ago, he describes as dull and aching in character, constant, precordial, steadily increasing, and radiating to the left scapula. He has cough for nine months, with expectoration and dyspnea on exertion, but no edema of the feet. Nycturia (1-2), but no pain, burning, or urgency. His sleep is at times disturbed by pain. He uses two pillows. I mention this because the number of pillows used often indicates roughly the degree of circulatory embarrassment from which an individual suffers.

He denies venereal infection, and questions regarding his habits and previous illness reveal nothing of medical importance.

You see before you a middle-aged man breathing rapidly, with difficulty, and, of necessity, propped up in bed. Orthopnea we call position, which indicates that the last line of circulatory and respiratory reinforcements has been called on. You can doubtless hear, even at a distance, his wheezing inspiration and short, choking, ineffectual cough, which suggests either a spasm or edema of the respiratory passages or pressure upon the trachea or bronchi. You notice the distended superficial veins of the arm, chest, and neck, which indicate a high venous pressure and an obstructed venous return flow to the heart.

The left pupil is much larger than the right. This condition may occur: (1) normally in some individuals, especially with refractive error, although I have never seen such a marked anisocoria thus produced; (2) from adhesions due to an antecedent iritis; (3) from pressure, producing an irritation of the cervical sympathetic.

But by far the most unusual and striking feature of the case is the bulging area of his chest, almost as large as half of a goose egg just to the right of the sternum at the level of the third rib. This area pulsates in an expansile manner, and upon palpation yields a systolic thrill.

I have drawn on the blackboard (Figs. 240, 241) a diagrammatic representation of the other physical signs.

Now, from what is this patient suffering?

A STUDENT: Aneurysm of the aorta.

DR. NORRIS: Yes, he presents practically all the typical late symptoms and signs. I can think of no other condition which produces an expansile pulsating tumor just above the heart. But let us examine his other signs more carefully. You will note that his heart is much enlarged, especially to the left. If this were due to a pericardial effusion rather than to left ventricular hypertrophy, his apex-beat would probably not be so dis-

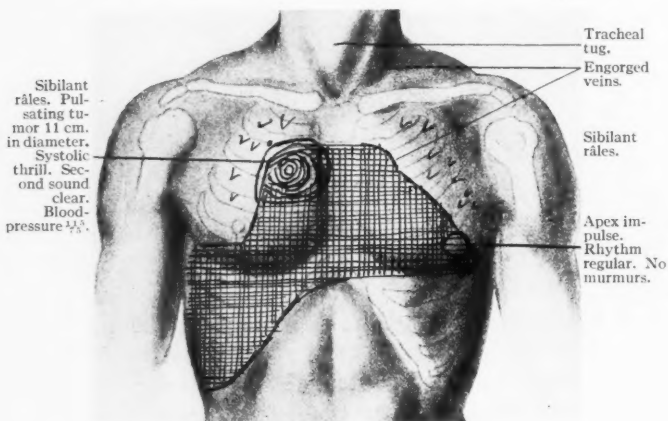


Fig. 240.—Physical signs anteriorly. Urine: slight trace of albumin, no casts. P. 80–100. Temp. 98°–100° F. Leukocytes 9520. Wassermann positive. x-Ray: aneurysm of aortic arch, large, and situated rather anteriorly.

tinct. Further, no friction sound is, or has been, heard. It is sometimes stated that cardiac hypertrophy does not occur in cases of aneurysm unless the valves are incompetent. Now this patient presents no murmurs, yet his hypertrophy is marked. One cannot, I think, afford to be dogmatic about such matters. He may have had arterial hypertension for a long time. Certainly hard labor entails temporary hypertension. It is generally believed that syphilitic mesaortitis plus hard work are the chief factors in the causation of aneurysm.

How shall we explain the dulness, bronchial breathing, and diminished tactile fremitus over the right lung posteriorly?

A STUDENT: Fluid in the pleura.

DR. NORRIS: Yes, and let me emphasize the fact, for it is too often a cause of error, that bronchial breathing and an increase of the whispered as well as the spoken voice sounds occurs in pleural effusion as well as in pulmonary consolidation. This is especially so in children in whom an empyema may be mistaken for a pneumonic consolidation. If the lung behind the effusion is solidified, either as a result of consolidation, compression, or

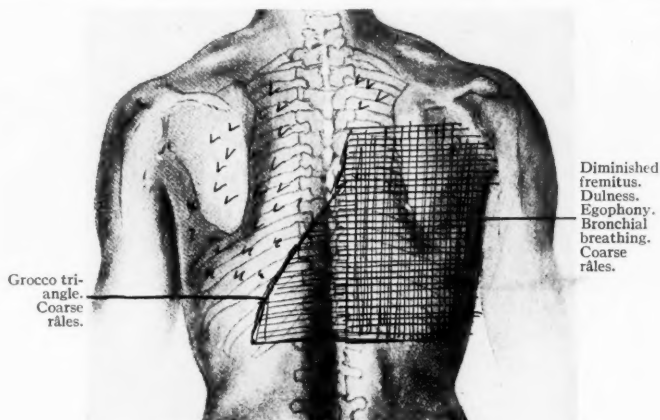


Fig. 241.—Physical signs posteriorly.

atelectasis, the signs of consolidation will be found. If, on the other hand, the lung be air-bearing and simply relaxed, breath sounds and voice sounds will be diminished or absent.

If you will look at this photograph (Fig. 242) you will see a good example of one of the types of late syphilitic aortitis.

*Microscopically.*—The chief lesions are perivascular infiltrations of the vasa vasorum, areas of small-celled infiltration in the middle coat, separation and degeneration of the elastica and the muscle-fibers.

*Macroscopically.*—The lesion is often localized to the arch

and does not extend more than 5 cm. above the valves. It is frequently characterized by an irregular, crinkled, thickening of the walls, giving a rubbery appearance, and at times showing

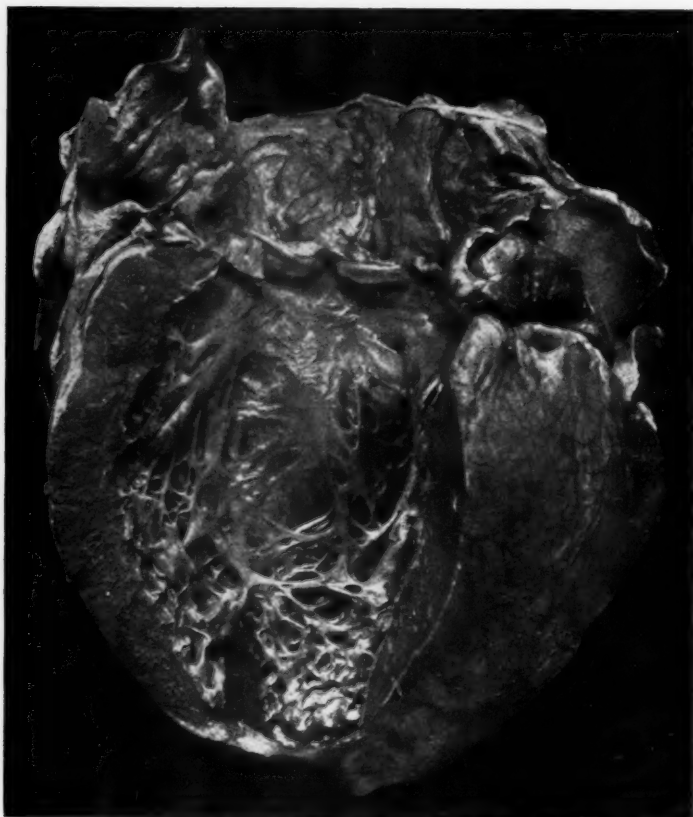


Fig. 242.—Syphilitic aortitis. Note the smooth, more or less circumscribed, rubbery appearance of the aorta and the thickened, shrivelled aortic leaflets. (From Norris' Cardiac Pathology, 1911, p. 203.)

small depressions or minute aneurysmal sacculations. The *Spirochæta pallida* can be demonstrated in the tissues. It has been truly said that "Venus loves the arteries" (Fig. 242).

I shall ask you to pass around these photographs from a very similar case of which I was fortunate enough, three years ago, to secure a set of frozen sections. They illustrate far better than could any description the intrathoracic relations in cases of this sort. You will find a descriptive legend attached to each (Figs. 243, 244).



Fig. 243.—Protruding aortic aneurysm, with bloody serum oozing through the skin. Death two weeks later by rupture into the left bronchus.

The patient depicted in the photograph was a negro stevedore, fifty years of age. He contracted syphilis in 1901, developed indigestion, pain and dyspnea in 1913, noticed a bulging of his chest in 1914. Received arsphenamine with temporary benefit in 1915, and died from rupture into the left bronchus in 1916.



Fig. 244.—Section through the thorax of the case shown in Fig. 241, cut through the middle of the chest, the spinal cord is seen cut in half. The manubrium has been eroded and absorbed. The sac is filled with laminated clot, only a small channel remaining open. At the posterior part of the latter rupture into the left bronchus occurred (*R*). The tracheal bifurcation (*T*) is filled with blood, as is also the esophagus (*E*) and stomach. The diaphragm and the liver are depressed. The heart displaced downward, both auricles are flattened. The roughened end of the sternum forms a part of the aneurysmal sac.

Another point should be explained—the hydrothorax. The patient has no edema of the legs or ascites. We cannot, therefore, assume a tricuspid insufficiency or anasarca from renal disease. The hydrothorax manifestly must result in both cases from pressure upon the pulmonary veins. You will note in Fig. 244 not only the downward displacement of the diaphragm, but the flattening of both auricles. The left auricle is pressed backward against the spine, and the ostia of the pulmonary veins are markedly compressed.

Now, gentlemen, we have seen today 2 cases illustrating the effects of syphilis upon the aorta. In the first case the damage extended downward, involved the valves, and produced a typical picture of aortic inefficiency, complicated by the symptoms of broken compensation, which occur when the myocardium begins to fail, the heart to dilate, and when the mitral and finally the tricuspid valves become inefficient. I wish to emphasize the importance of the myocardium. The French express this very well in saying, "A disease of the valves is not a disease of the heart." Illustrations of this are seen in young adults and in experimental animals, where valvular damage is, as a rule, unimportant so long as the heart muscle is young and vigorous. There are but few lesions, mostly congenital, which cannot be, and in young persons usually are not, compensated to a degree which is perfectly compatible with a useful, comfortable, and unrestricted existence.

The second case illustrates an upward spreading of the specific aortitis with gradual weakening and rupture of the medial coat, and finally the development of a large aneurysm.

The diagnosis of both of these cases is easy and obvious. What I would like to impress upon you is the desirability of ever bearing the possibility of an aneurysm in mind, especially in or beyond middle life, in any patient who complains of chest or back pain, dyspnea, or cough which does not yield to treatment, or for which a definite cause cannot be found. I can assure you that by so doing you will spare yourself many a humiliation.

**Treatment.**—Aneurysm is, of course, incurable. All that we can hope to do is to check or to retard development and to alle-



Fig. 245.—Aneurysm of the aorta which has been "wired," showing wire and clot.

violate suffering. Inasmuch as nearly all thoracic aneurysms are syphilitic in origin, mercury, iodids, and arsphenamine are our sheet-anchors. If the aneurysm lies anteriorly, "wiring" may



be tried. This procedure consists in thrusting many feet of fine gold wire into the sac, and then passing an electric current through the wire in order to produce clotting. This often relieves pain and prolongs life. It is an operation, however, which requires a very special technic and skill. Further, there is always the possibility that part of the clot may become detached and sudden death occur during the operation. The photograph I have here depicts an aneurysm which has been wired (Fig. 245).

If pulmonary edema occurs, the patient should be promptly and profusely bled. In one case under my care this procedure not only saved the life of a patient who seemed moribund, but enabled him to leave the hospital in a condition of comfort. He returned two months later with a recurrence of symptoms and died about two weeks after his second admission.

Aside from wiring, bleeding, and rest in bed, treatment is purely symptomatic. A hydrothorax may be aspirated, with temporary relief. Opiates are usually required in the late stages and should be freely administered.

I have placed upon the blackboard some statistical compilations taken from Lemann's article, showing age incidence, incidence of symptoms, and site of rupture in a large series of thoracic aneurysms.

AGE INCIDENCE OF THORACIC ANEURYSM (LEMANN)

Age, years.	Lemann's Touro series.	Lemann's Charity Hospital autopsy series.	Bassett Smith.	Maxim- off.	Maximoff from literature.	Borowsky from literature.	Crisp quoted by Borow- sky.	Emerich quoted by Borow- sky.
1 to 10...	...	...	...	...	10	1	1	
10 to 20...	...	...	...	...	9	2	5	
20 to 30...	1	6	13	4	28	11	71	1
30 to 40...	15	17	28	4	71	43	198	7
40 to 50...	19	20	6 over	14	100	58	129	16
50 to 60...	8	9	...	14	56	46	65	16
60 to 70...	3	3	...	5	21	10	25	11
70 to 80...	...	...	...	...	5	4	8	5
Over 80...	...	1	...	...	4	..	3	2
Unknown..	1	10						

You will observe that the majority of the cases occur between the ages of twenty and fifty years. This is, of course, earlier than the time at which arteriosclerotic phenomena usually manifest themselves.

As will be noted in the following table, the most constant physical sign is substernal dulness, while chest pain constitutes the most frequent symptom.

#### INCIDENCE OF SYMPTOMS: TOURO CLINIC SERIES

	Present in cases.	Absent in cases.	Not men- tioned in cases.
Bruit. ....	20	9	18
Tracheal tug. ....	4	16	27
Substernal dulness. ....	33	3	11
Vertebral dulness. ....	15	5	27
Inequality of pupils. ....	4	25	18
Inequality of radials. ....	5	21	21
Unequal sweating. ....	0	0	47
Left recurrent paralysis. ....	6	2	39
Cough. ....	16	5	26
Pain in chest. ....	27	..	20
Pain on pressure over sternum. ....	4	..	43
Pain in right arm. ....	1	..	46
Pain in left arm. ....	7	1	39
Dysphonia. ....	9	1	37
Dysphagia. ....	7	1	39
Diastolic shock. ....	4	14	39
Tumor. ....	6	7	34
Thrill. ....	4	12	31
Pulsation. ....	10	1	36
Wassermann. ....	11	9	27
Tchergunobow modification of Wassermann. .	10	5	27
Fluoroscope. ....	12	..	35
Roentgenogram. ....	32	1	14
History of syphilis. ....	11	3	33

Practically all aortic aneurysms are a late result of syphilitic aortitis, and about 30 per cent. of all cases of the last-named lesion ultimately develop into aortic aneurysms. The average length of time elapsing between the aortitis and the aneurysm is about sixteen years. The onset of aneurysm may, however, occur within three years or be delayed as long as forty-one years.

The belief currently held that the average length of life after

the diagnosis has been established is about two years is borne out by Lemann's figures:

3 months.	4 cases.
3 to 6 "	12 "
12 to 15 "	17 "
15 to 18 "	10 "
18 to 24 "	14 "
24 to 36 "	10 "
36 to 48 "	8 "
4 to 10 "	9 "

The following table shows the site of rupture in a series of 603 cases. It will be noted that nearly one-quarter of the cases rupture into the pericardium, the left pleura being the next most frequent location.

## RUPTURE (603 CASES)

		Per cent.
Pericardium.....	150	24.0
Left pleura.....	101	16.0
Esophagus.....	53	8.7
Trachea.....	51	8.4
Right pleura.....	45	7.4
Left bronchus.....	38	6.02
Externally.....	35	5.6
Superior vena cava.....	31	5.1
Left lung.....	23	3.08
Pulmonary artery.....	18	2.09
Mediastinum.....	11	1.9
Right lung.....	10	1.6
Right bronchus.....	8	1.3
Elsewhere (right and left auricles, left ventricles, etc.)....	25	4.1

I do not wish, however, to leave you under the impression that rupture is the commonest cause of death. It occurs in about 40 per cent. of the cases.

As has been illustrated by the cases you have seen and as is borne out by statistical studies, syphilitic aortitis occurs relatively early after the initial lesion. If the cases are inadequately treated or not treated at all, about 30 per cent. of them will develop aneurysm about twenty years later. This lesion occurs chiefly in males (80 per cent.), especially in those who perform

heavy labor which entails lifting and straining. Here in Philadelphia negro stevedores are notoriously subject to aneurysm. About 15 per cent. of the cases of syphilitic aortitis develop a dilatation of the aorta without actual aneurysm, and about 30 per cent. result in sclerosis and retraction of the aortic leaflets. Those of you who may care to investigate the subject of aortic aneurysm more thoroughly should look up the following articles:

The Etiology of Syphilitic Aortitis, Symmes, David Wallace, G. H., Jour. Amer. Med. Assoc., 1916, lxvi, 397.

Aneurysm of the Thoracic Aorta, its Incidence, Diagnosis, and Progress. A Statistical Study, Lemann, I. I., Amer. Jour. Med. Sci., 1916, clii, 210.

Syphilitic Aortitis, its Diagnosis and Treatment, Longcope, W. T., Arch. Int. Med., 1913, xl, 15

The Treatment of Aneurysm of the Aorta by the Introduction of Wire and the Passage of a Galvanic Current, Eshner, A. C., Amer. Jour. Med. Sci., Oct., 1910.

CLINIC OF DR. JOHN H. MUSSER, JR.

UNIVERSITY HOSPITAL

THREE INSTRUCTIVE CASES

**Case I: Aortic Aneurysm. Case II: Pericarditis and Endocarditis. Case III: Aplastic Anemia. Histories, Diagnoses, Treatment, and Management.**

CASE I.—AORTIC ANEURYSM

THE first case which I wish to present to you is that of aneurysm of the aorta. It presents some interesting features from a diagnostic and, more particularly, from a therapeutic standpoint. The history of the patient briefly is as follows:

W. W. White, male, age forty-three; occupation, waiter. Admitted August 28, 1919 to Philadelphia General Hospital.

**Chief Complaint.**—Pain in left chest—most severe beneath inferior angle of scapula.

**Present Illness.**—About one year ago began to have attacks of pain between shoulders and over sternum. Attack would come on after heavy day's work. The patient would take four or five drinks of whisky and go to bed; in the morning pain was relieved. The attacks have gradually gotten closer together and have persisted over longer periods of time. The present attack began about two weeks ago and is the worst that the patient has had. The pain is sharp—cutting—causes difficulty in breathing. At present most severe pain is around inferior angle of the scapula on left side. Patient has difficulty in swallowing; swallowing also causes considerable pain.

**Family History.**—Father's whereabouts unknown. Mother died of tuberculosis at age of thirty-four; one brother, unknown as to present condition; one sister living and well.

**Past Medical History.**—Always healthy until one or two years ago. Has had gonorrhea and chancre (soft?) at age of seventeen. He was subsequently circumcised, and later took treatment at Hot Springs, Arkansas.

**Habits.**—Patient has always been a heavy drinker of whisky and a heavy smoker of cigarettes. Uses no drugs. Climbed many stairs daily while working.

**Physical Examination.**—Slightly emaciated white man of about forty-three years of age. *Eyes* and pupils unequal and react to light and accommodation sluggishly. *Nose:* Perforated septum. *Mouth:* Tongue heavily coated; fine tremor at tip. Has an upper plate; lower teeth in bad condition. *Neck:* enlarged postcervical glands; no tracheal tug. *Chest:* Prolonged expiration over right apex and extending down to fourth interspace. This same area gives increased fremitus, but no râles. *Heart:* Apex barely palpable in fourth interspace just inside midclavicular line. To right of the sternum in second and third interspace and extending 7 cm. to the right of sternum is an area of expansile pulsation. The expansion is synchronous with the pulse-beat. Heart sounds are very distant over cardiac area, but over the pulsating area to the right of the sternum the sounds are very distinct, showing a soft, blowing systolic murmur at the end of systole and a very snappy ringing second sound. *Abdomen:* Negative. *Reflexes:* Normal. There is a slight difference in time of occurrence and volume of the two radial pulses.

**Discussion.**—The *diagnosis* in this case was, of course, relatively easy. The patient has a large pulsating protuberant mass in the right chest just to the outer side of the sternum. It would have been very difficult to make any other diagnosis than that of aneurysm. Other pulsating tumors might have produced a similar picture in the chest wall, but such a tumor would not have been expansile nor would there have been asynchronous radial pulses or a snappy, ringing second sound. The diagnosis of aneurysm was entirely substantiated by the Roentgen ray.

In regard to the *etiology*, the patient not only gives a history

of having had syphilis but also had a strongly positive Wassermann. Did we not know at the present time syphilis is the almost universal cause of all aneurysm, we could show other factors in his history which, ten years ago, might have been held to be of etiologic importance. For example, the man was a heavy drinker, climbed many stairs while working, and had other etiologic causes which were formerly frequently held responsible for this condition.

In this patient these factors undoubtedly played an important part in his present condition. The arterial wall was weakened by the spirochetal invasion of the media, and it is to be borne in mind that syphilis of the arteries commences as a mesaortitis. The main protecting coat of the artery was diseased. It was subjected to rather violent strain of increased blood-pressure during the frequently recurring drinking bouts and also to the increase of blood-pressure that accompany heavy physical effort—in this case the effort required to carry a heavy load of dishes up several flights of stairs. Finally, under the repeated strain, one small section of the media gave away; constant but irregular pressure caused a weakening of the wall, which gradually enlarged, and an aneurysmal sac was formed.

The **treatment** of aortic aneurysm in general and this type of aneurysm in particular is what I wish to lay particular stress upon. In view of the strongly positive Wassermann reaction intensive treatment to eradicate the syphilis is indicated. The rule of lues in the etiology of this condition is so important that antisyphilitic treatment is practically always necessary. Treatment for the aneurysm *per se* is, of course, extremely difficult. Various treatments have been elaborated in order to promote clotting of the blood within the sac, with subsequent organization. Some of these have for their purpose the increasing of the viscosity of the blood by drugs or by physical means, such as reduction of fluid intake, dry diet, and so on. In a certain number of selected cases fine wire has been introduced into the aneurysm and a galvanic current applied to the wire. The purpose of electrolysis is the localized formation of a clot within the sac of the aneurysm, which would become firmly organized and prevent

further enlargement and gradual erosion of the aneurysm through the surrounding tissue. The wiring of aneurysm was first performed by Moore in 1864; fifteen years later Burres combined wiring and electrolysis. In a certain number of cases this method has yielded brilliant results. Hobart A. Hare of this city is enthusiastic over this method of treating aneurysm, and has reported some very good results. The indications of procedure are, first, rapid erosion of the aneurysm; second, excessive pain; third, a well-formed sacculated aneurysm with a comparatively small stoma. In the present patient it was thought advisable to wire the aneurysm because of the severe pain the man was having and because the fairly well-developed sacculated aneurysm offered the possibility of having a clot form within the sac, adhere to the vessel wall, and by increasing its strength prevent further damage. The wiring of the aneurysm was done on September 30, 1919. A very fine gold wire was used, and a needle of small caliber, but sufficiently large for the passage of the wire, was also employed. The needle should be covered with white enamel and absolutely smooth and without irregularities, so that it will cause the minimal trauma to the arterial wall. The needle was introduced in the third interspace just to the right of the sternum, slipped in quite easily, and as it progressed inward the pulsation of the aneurysm could be felt very readily. Slightly increased pressure forced the needle through the aneurysm wall and the dripping of blood from the proximal end showed that it had gone in safely. The wire was wrapped on a spool so that it would curl when it got into the aneurysm. The wire, unfortunately, was not entirely satisfactory, and whereas I had planned to introduce 20 feet, I was only able to get in 12 feet on account of the wire breaking several times, necessitating the withdrawal of the wire already in the sac. The last 12 feet were introduced slowly and the galvanic current applied. It is advisable to have the galvanic current applied to the end of the wire protruding from the chest wall with the other electrode applied to the muscles of the back. The strength of current should be rather rapidly increased to between 40 to 50 milliamperes, and should be applied for twenty to



thirty minutes. At the conclusion of the galvanization the small end of the wire still protruding from the chest wall is pushed in as far as possible and the puncture closed with collodion. The operation was quite prolonged due to inability to introduce more



Fig. 246.—Roentgenogram taken Dec. 1, 1919. The wire in the sac of the aneurysm may be faintly seen to the right of the sternum. Note also enlargement toward the left, absent in the earlier roentgenograms.

than a small amount of wire at a time. While the needle was in the aneurysm the heart became absolutely irregular; apparently the auricles were thrown into fibrillation; except for this untoward event nothing of moment took place during the operation.

The patient was kept in bed flat on his back for four days. He was told to avoid all strain of any kind. Irregularity of pulse disappeared shortly after his return to the ward. In a note that Dr. Costello has made October 1st he states that the severe pain in the shoulder-blade has entirely gone; October 4th he also notes that pains of all kinds have about gone except the abdominal distress and the feeling of restriction across the chest; October 5th the patient sat up for the first time; October 19th he was out of bed. On November 11th a note was made that the patient fears the old pain is returning; today, as you see, the man has comparatively few complaints. He has attacks of dyspnea upon slight exertion; he has pain at the present time which is not as severe or as sharp as before. His pain starts to the right of the sternum and is felt down the right arm. He says it is rather sharp and paroxysmal and is easily brought on by some slight exertion. The examination shows that the expansile protuberant mass is still present, but has subsided considerably. There is a distinct impulse which can be readily seen in the third and fourth interspaces. The dulness to the right of the sternum extends down several centimeters below the previous level of dulness. It would seem that now the aneurysm is enlarging downward and becoming more fusiform in shape. This will be confirmed or disproved by the x-ray.

#### CASE II.—PERICARDITIS AND ENDOCARDITIS

The person I wish to present to you today is a young man whom I treated at the Presbyterian Hospital some five years ago. I want to read his history at that time and his physical condition which was present then and compare it with the present conditions. The patient is a young man with an enormous enlargement of his heart. After we have gone over the history I wish to give you all the opportunity of palpating the precordium and listening to the heart sounds. The case presents nothing of particular difficulty in diagnosis, but is an interesting evidence of a severe degree of cardiac pathology which we sometimes see in persons who are apparently able to go on with their usual daily occupations. Mr. W. does not come here today to consult me

as a patient, but has kindly come down here to give me the opportunity of demonstrating his heart to you.

**History.**—In 1914, about the last of November, he entered the Presbyterian Hospital on account of pains in his knees and over his heart, as well as extreme dyspnea. This precordial pain and distress developed about two weeks before his entrance into the hospital. This, together with the pain and swelling of the knees, became so severe that he was obliged to go to bed four days after being taken sick. He had a sore throat the first few days of the sickness, and this was followed by pain and swelling of the knees. Mr. W. has had frequent attacks of rheumatism and tonsillitis. He has also had measles and mumps and a bronchopneumonia when two years of age. In the family history we note that his father died of tuberculosis, and his mother, three brothers, and three sisters are all living and well. His occupation is a stenographer, his work is not particularly difficult for a boy of eighteen, and he has no bad habits. From this brief history, taken five years ago, we note, in brief, that there has been a history of repeated attacks of rheumatism induced by a sore throat in all possibility, the tonsils being the portal of entry for the invading organisms. In all probability when he was first taken sick pathogenic bacteria got into his blood by way of the tonsils and induced the endocarditis.

The notes on his examination at this time are briefly as follows: The patient lies in a dorsal decubitus, with knees drawn up, and in apparent distress from extreme dyspnea, each respiration beginning with an effort. Nutrition is fair, but skin is pale and moist. There is no engorgement of the superficial veins and no definite edema. Examination of the head shows pallor of the mucous membranes and conjunctiva. The teeth are in good condition, tongue dry and slightly coated. Tonsils show evidence of chronic inflammation. They are rather small, irregular, and there would appear to be thin adhesions to the posterior pillars. The neck shows no enlargement of the lymphatics, but anteriorly there is a marked pulsation of both carotids and jugulars. The thyroid is negative. The examination of the chest shows the left side more prominent than the right; expansion

of the chest is limited and jerky; apex-beat is wide and diffuse in the sixth interspace, anterior axillary line; area of cardiac dullness is greatly increased; right border is 1 cm. to the right of the right border of the sternum; left in anterior axillary line in the sixth interspace; the upper border at third rib. The cardiohepatic is acute. Over the heart there is heard at the apex a high-pitched whispering murmur transmitted into the axilla. This is followed by a very low, short diastolic murmur. Over the whole precordium a two-and-fro friction-rub is heard with each heart-beat. The area of maximum intensity is at the left fourth costal cartilage. At all the valve points the heart sounds are almost obscured by the murmurs. The lungs show at the base of the left scapula a few râles and prolongation of the expiratory note. Examination of the abdomen shows the liver is slightly enlarged downward, is not palpable, but distinctly tender. The abdominal examination otherwise is negative.

Two days after admission to the hospital the patient developed a sharp rise in temperature, up to 102° F. With this the pain practically disappeared and the dyspnea was considerably improved. In the left axilla a friction-rub, pleuropericardial, developed. Four days later we note that physical signs on the left interior angle of the scapula are very similar to those that are heard when there is a solidification. We also note that he has, for the first time, developed an aortic diastolic murmur. The area of cardiac dullness is extended 4 cm. to the right of the sternum. The apex-beat is still heaving. The obliteration of the cardiohepatic angle, the disappearance of the precordial friction-rub, the enlargement of cardiac dullness at the base of the heart to the right of the sternum, and physical signs at the left base of the lung induce us to do a paracentesis. Needle was inserted to area of fourth interspace 2 cm. to the right of the sternum; 400 c.c. of a serosanguineous fluid was aspirated from the pericardial sac. The patient was very much relieved of his dyspnea. The cardiohepatic angle on the following day had become obtuse. The right base measured 8.5 cm.; left, 15 cm. The examination of the pericardial fluid showed 9600 leukocytes per cubic millimeter and the biuret test was positive. The

leukocytes with the rise in temperature had gone up to 34,000; they gradually fell to 19,000 five days after the paracentesis, of which 90 per cent. were polynuclears.

From then on convalescence was good. A small fluid collection was noted at the right base and a small quantity of sterile fluid withdrawn. Shortly before his discharge the right base measured 5 cm.; left base, 13.5 cm. He was discharged February 2d in fairly good condition. Laboratory examination in the hospital showed from time to time traces of albumin in the urine,

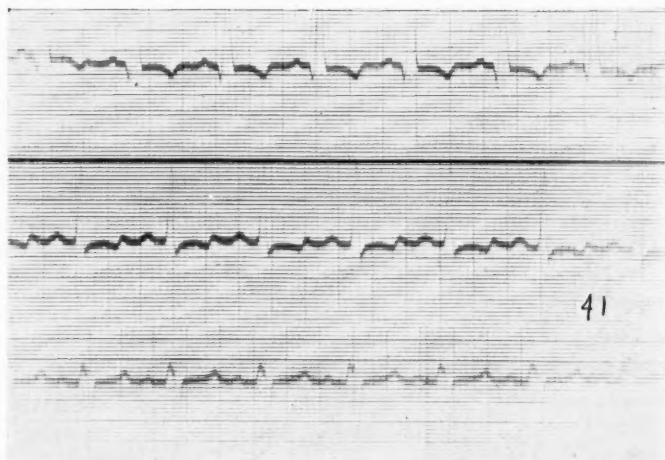


Fig. 247.—No. 41. Pulse-rate 120. Left preponderance. Note the similarity between this electrocardiogram taken Jan. 25, 1915 and the one taken four years later, Nov. 25, 1919.

specific gravity of which was usually high; occasionally casts were seen. There was a fairly well-developed secondary anemia; phthalein elimination was 38 per cent. the first hour, 15 per cent. the second. Electrocardiograph taken at the time showed a well-marked left preponderance. Mr. W. was readmitted to the hospital fifteen months later with an arthritis. The course of this admission was comparatively short and he was discharged in a few weeks. There was no apparent change in the heart at this time.

At the present time Mr. W. is apparently free from subjective symptoms. I saw him a few days ago in my office, and he had walked rapidly at that time a distance of about eleven squares without any subjective inconvenience. Today, as you see, he is not dyspneic or uncomfortable, although he has hurried here from the place where he works. He tells me that at times he notes some palpitation, but, on the whole, this symptom bothers him very little. This is really quite surprising. When you see the size of his heart and its vigorous action you will wonder why he does not have more symptoms. He has gained some weight and in every respect he is apparently normal. Although he has so few subjective symptoms, you will see his objective symptoms are much more pronounced. We will get him to take off his clothes in order to examine the heart more carefully. You will now be able to see more fully the marked changes that the injury to the heart has brought about. You will see that Mr. W. is a small, slight, undernourished individual. The pulsations in the neck are quite marked; not only the carotid artery pulsates, but there is also a pulsation above the sternum. The whole left side of the thorax is bulging out, and I should say that it projected at least 6 cm. further outward than does the left side. The whole left side of the chest is being thrust out with each cardiac impulse, and in view of the general pushing out of the chest wall you will observe that there is a rippling impulse which starts at the third interspace and runs into the axilla, and that there is also a localized impulse to the right of the sternum. There is also retraction in the region of the eighth costal cartilage. On palpation of the precordium we note this marked heaving impulse, and we also notice a thrill at the apex. The heart rate is also increased. On persussion of the heart we note the following: the area of cardiac dulness extends from the upper border to the second rib at its furthest point 4 cm. to the right of the sternum, and the left 16 cm. in the fifth interspace, although the dulness extends down to the sixth interspace in the midaxillary line. You see we have a small undersized male, weighing only 115 pounds. This is a perfectly enormous organ. On auscultation we can hear practically nothing except murmurs

at each valve area as well as over the entire heart. The heart sounds are practically entirely replaced by these murmurs, which are both systolic and diastolic in time. There is no

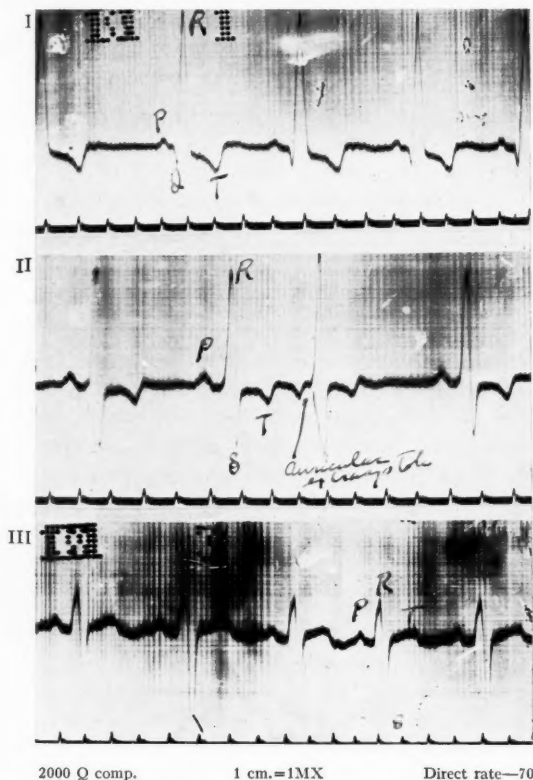


Fig. 248.—Left ventricular preponderance. One auricular extrasystole in Lead II. Inverted *T* in Leads I and II if not due to digitalis probably evidence of myocardial weakness. (Electrocardiogram made by C. C. Wolferth.)

irregularity of the heart. The aortic second sound is possibly slightly accentuated. Electrocardiograph, taken at this time, shows much the same findings as the one taken four years ago.

**Discussion.**—I wish now to say a few words concerning the diagnosis of pericardial effusion. There are a few physical signs which to me seem to be of particular value. In the first place the area of dulness is a rather peculiarly pear-shaped form, and with it there is obliteration of the cardiohepatic angle. This I consider of particular value. In larger effusions there are signs at the angle of the left scapula, particularly noteworthy in this case. I cannot fail to see why this is not due to compression of the lung, because we have here, for example, a very large precordium with a large amount of fluid material in it which is continually pressing backward on the lung; certainly the physical signs are those of a slight solidification from compression. Another important sign is the tendency for the heart sounds to be muffled. Theoretically the apex-beat is supposed to be obliterated, and while this is given as a classical symptom in most text-books, I have never had the opportunity to see it. On the contrary, in all effusions that I have noted there always is a marked impulse over the precordium.

**The Course.**—As a result of this pouring out of the fluid in the pericardial sac there is no doubt whatever that the epi- and pericardial layers of the sac become adherent one to the other. You are aware, also, that in the history the pleuropericardial rub was heard, so that there is a strong possibility that the pericardium may be adherent to the chest wall. There is, however, no retraction of the chest wall during the systole of the heart that occurs when there is an obvious well-marked attachment of the pericardium to the pleura. This adhesive pericarditis, as it is called, throws an extra burden upon the heart. The heart does not have the smooth, well-lubricated surface of the pericardium to work within, instead of which there is more or less a restraining membrane upon it.

The mechanical obstruction to the heart in this patient is not so marked as we see it when we have a gross pericarditis with adhesions to the mediastinum, which frequently is associated with enormous increase in the size of the heart, cirrhosis of the liver, and ascites (Pick's disease). We see now that in addition to the heart lesions we also have the complication of



this fibrous pericardium, all of which calls for additional work by the heart. How long the heart will be able to keep this up is impossible to say. There is no question that it has responded very splendidly during the past four years, however, with a well-marked increase in the hypertrophy. How long the cardiac muscle will continue to hypertrophy is another question, because in all likelihood the limit of enlargement has been about reached, and the subsequent history will be dilatation with signs of cardiac insufficiency. In the present discussion we have gone over the pericardial condition rather fully. I do not think that there are physical signs at present to diagnose this lesion did we not know the previous history. The most obvious physical sign is the retraction at the eighth costal cartilage which is frequently observed during the course of chronic pericarditis.

Now a word as to valve lesions. There are systolic and diastolic murmurs at each valve area. These may be murmurs which arise at other valve areas, but it is more likely that at each valve there exists not only a relative insufficiency but also a relative stenosis. The heart has become so greatly hypertrophied that it is impossible for the valves to respond to this great enlargement of the cardiac cavities. The valves are stretched about to their limit of distensibility, and the valves consequently during systole close incompletely, leading to an insufficiency of the several valves. Likewise the cavities of the heart have become so great that each valve has become relatively small for the size of the cavity, and, in turn, has become stenotic because of its size as compared to the larger cavity. Thus we have not only a relative insufficiency but also a relative stenosis. In this connection I might call attention to the fact that during his previous sickness the signs of aortic insufficiency developed. The aortic ring (so called) is extremely powerful, and this valve is almost always the last valve to be affected by a relative insufficiency. There is just one other point I wish to call attention to, and that is, that in spite of the valvular insufficiency the tricuspid shows very little signs of insufficiency as shown by the changes in the liver. This is to be accounted for by the fact that the major

part of the condition is developed around the left heart, while the right heart has played a comparatively small part.

The **treatment** of this patient may be summed up in a word—hygiene. He should live an absolutely perfect hygienic life, with complete avoidance of anything that will throw extra work upon the heart. There is no indication for stimulation or for any drugs. Later on, when the cardiac insufficiency develops, then we will have recourse to our drugs.

#### CASE III.—APLASTIC ANEMIA

The third case I wish to report to you briefly is one in which the patient is now dead, but which, as I have the opportunity of showing you the bone-marrow, it might be well to discuss the clinical aspects of the disorder. This patient was on Dr. Joseph Sailer's service at the Presbyterian Hospital where I had the opportunity of studying her. She had been a maid in the hospital wards for some sixteen odd years and had had no particular illness during her service there. Finally, last spring she was compelled to enter the hospital on account of increased weakness, and for which she had not consulted any of the hospital staff before her admission to the ward. Her history, briefly, is as follows:

D. G., aged 61; colored; occupation, maid. Admitted to the Presbyterian Hospital April 21, 1919.

**Chief Complaint.**—Shortness of breath and general weakness.

**History of Present Illness.**—The present attack commenced about five or six weeks ago with shortness of breath and a general feeling of weakness. This has grown progressively worse. Patient was forced to give up work six weeks ago because of this weakness. Believes she has lost about 20 pounds during this period. Associated with the weakness there have been attacks of shortness of breath, with some palpitation. At times there has been a sharp, fleeting pain in epigastrium. This has generally disappeared after meals and has been of short duration. There has been a good deal of belching of gas, this usually occurring in the evening. There has been no headache; appetite has

remained good. Bowels would be constipated except for medicine which she has taken. No night-sweats. No cough. Menopause about ten years ago. No symptoms since. No genito-urinary symptoms. An attack of shortness of breath and weakness such as this was quickly relieved about a year ago.

**Past Medical History.**—Several attacks of articular rheumatism during past four years. Last attack, of a mild degree, was this past winter. Elbow-, knee-, wrist-, and ankle-joints have been affected. No history of associated endocarditis. A good deal of throat trouble and two attacks of pleurisy in youth. Menstrual history started at fourteenth year, lasting four days, regular, flow normal. Otherwise negative.

**Family History.**—Mother died about forty, of "lung trouble"; father, about fifty-five, of Bright's disease. Second husband died of typhoid fever, first one of pneumonia. Two children by first marriage, living and well.

**Social History.**—Patient has been employed in general work about this hospital for past sixteen years. Home conditions good.

**Physical Examination.**—Patient is an elderly, poorly nourished female negro, of markedly sallow appearance. Skin is mottled and so pale that she has the appearance of a white woman.

**Head.**—Scalp, ears, nose, negative. Eyes, negative; moderate degree of exophthalmia; sclera and conjunctiva of an icteroid appearance; pupils equal and moderately contracted; react to light and accommodation; extra-ocular movements normal.

**Mouth.**—Tongue and mucous membranes pale; teeth poor; throat, very slight congestion.

**Neck.**—No adenopathy. Thyroid is not visibly enlarged; no venous pulsation.

**Chest.**—Expansion fair and equal; vocal resonance normal; negative to auscultation and percussion both anteriorly and posteriorly.

**Heart.**—Apex-beat is prominent in fifth interspace, mid-clavicular line. Area of heart dulness slightly enlarged to left.

Systolic murmur is heard at apex. It is not transmitted to axilla. Sounds over aortic cartilage are negative. No areas of pulsation visible or palpable beyond that of apex-beat.

*Abdomen.*—There is an area of tenderness in midepigastrium just below xiphoid. No mass is palpable at this point. Liver and spleen are not palpable. No other areas of tenderness. Abdominal wall is relaxed and note tympanitic throughout. No edema is present.

*Back.*—No areas of tenderness along vertebral column or in sacro-iliac region.

*Extremities.*—*Upper:* Reflexes present and normal; matrix of nails is quite pale; no adenopathy. *Lower:* Reflexes present and normal, no Babinski, no ankle-clonus. Both ankle-joints are somewhat enlarged as if from chronic joint inflammation. There is some slight suggestion of pitting on pressure along tibia of both legs. Otherwise negative.

*Laboratory Reports.*—*Urine.*—Repeated examinations of the urine showed four times traces of albumin and occasionally hyaline and granular casts.

*Stools.*—Five stool examinations showed nothing abnormal.

*Stomach Contents.*—There was an absence of free HCl, with a total acidity of 9.

*Blood.*—On admission the count was very low, Hg. —16 per cent., red blood-cells, 720,000; white blood-cells, 2700. Immediately following transfusion there was a rise in hemoglobin to 22 per cent., and red cells to 1,550,000. This was followed by a gradual rise for eight weeks, at the end of which time the hemoglobin was 52, red cells 2,280,000, and white cells 4900. The count again gradually fell until August 12th, when the hemoglobin was 37 and the red cells 1,990,000. The white count was 4200, of which 63 per cent. were polynuclear cells, 31 per cent. lymphocytes, 1 per cent. large mononuclears, and eosinophils 5 per cent. This was the highest figure the polynuclears and eosinophils reached. In all the other differential counts that were made the lymphocytes were greatly increased relatively. After a transfusion the erythrocyte count was 3,460,000 and hemoglobin 45 per cent. Fifteen days later the red cells had fallen

to 1,930,000. There was a slight rise the next two weeks, but they persistently fell, and each weekly count was lower than the previous one until November 10th, when the red cells were only 880,000 per cubic millimeter and the hemoglobin 22 per cent. A suitable donor was at last secured and the count was increased by transfusion by about 500,000 cells and the hemoglobin 10 per cent. By December 1st the count was: erythrocytes 980,000, leukocytes 1650, hemoglobin 24 per cent. The remaining counts were about the same, the last one shortly before death, on December 29th, being 850,000 and 22 per cent. Erythroblasts were never seen and there was little change in the general appearance of the red cells, though they were apparently smaller than normal. The Wassermann was negative.

I will now read a summary of the autopsy report made by Dr. John Eiman:

Summary of autopsy findings on Mrs. D. G., 1919. Body is that of a poorly nourished adult colored woman weighing about 50 kilos. The musculature is very poor, panniculus adiposus very small amount. Skin is loose, dry, and brownish yellow in color. The scleræ are yellowish, the mucous membranes are almost white. There is slight edema around the ankles. Thoracic and abdominal muscles are rather bright red. There are rather numerous subpleural and subperitoneal petechial hemorrhages. Lungs and pleuræ show no special features. Heart shows extreme degree of fatty degeneration. Liver weighs 1020 grams. The edges are sharp, consistency is somewhat decreased. Cross-section is brown in color, liver lobules are indistinct.

*Sections.*—Capsule not thickened, interlobular connective tissue is not increased. Hepatic cells are not well defined; they show moderate amounts of brown pigment especially toward the middle of the lobules. Koffer cells are charged with similar pigment.

*Spleen* weighs 30 grams; the capsule is irregularly thickened, consistency is increased. Section surface is brown in color, trabeculæ are prominent, follicles are indistinct.

*Sections* show thickened capsule. Trabeculæ are rather dense. Malpighian bodies are hyalinized. Intersinusoidal tissue of

pulp is decreased. Endothelial lining of sinuses is swollen and there is proliferation of endothelial cells. Sinuses contain large numbers of desquamated endothelial cells. Lymphoid elements of pulp are diminished. Atrophy of spleen with endothelial hyperplasia. Kidneys show cloudy swelling and deposits of a brownish pigment, especially in convoluted tissues. Stomach and intestines show numerous petechial hemorrhages. There is no apparent atrophy of the mucous membrane. Bone-marrow of tibia: the spongy portion is yellow in color, the marrow cells are entirely replaced by fat. In the shaft the marrow is yellow in color, with an occasional reddish streak. Sections show variations from ordinary fat in fields, in which the capillaries between the fat cells are prominent, to occasional areas in which the fat occurs as isolated cells, separated by small islands of marrow cells. Cellular marrow consists mostly of myeloblasts and myelocytes, with early forms of polymorphonuclears. Differentiation between myelocytes and myeloblasts cannot be made with certainty. There are also numbers of hemoglobin red cells appearing both as myeloblasts and myelocytes; occasional cells show double skeins, indicating multiplication. A few of the larger leukocyte-forming elements show coarse eosinophilic granulation.

Though the marrow shows some activity, the relative amount of the cellular marrow is so small that the diagnosis of pernicious anemia of the aplastic type seems justifiable.

**Discussion.**—This was a case of aplastic anemia and presents a somewhat different type from the case I reported from the same sources a few years ago (*Arch. Int. Med.*, 1914, xiv, 275). At that time I gave the definition of "aplastic anemia" as a type of anemia different in many respects to the ordinary types of primary or secondary anemia, marked especially by retrogressive changes of the bone-marrow which result in the change of the normal red marrow to a fatty marrow. Comparatively few cases have been reported, but it seems this is due more to the fact that these cases have been unrecognizable or improperly studied rather than to their rarity. The condition occurs usually in early adult life, and is distributed about equally between males and females. The course of the disease is, as a rule, rapidly

progressive without the customary remissions which are observed in pernicious anemia. Another unusual feature of the disease is the marked tendency to subcutaneous hemorrhage or hemorrhage from the mucous membrane. Fever is also a common symptom. The blood-picture is quite characteristic. There is usually a rather severe and intense anemia of the hemoglobin and the red cells, with a low color-index, though in a well-studied case recently reported by O'Malley and Conrad (Jour. Amer. Med. Assoc., Dec. 6, 1919, p. 1761) the anemia was relatively slight. The red cells show but little change in their morphology. There is, in addition, leukopenia, which is well marked. There is a relative decrease in all types of the leukocytes except the lymphocytes, which are relatively increased.

At postmortem the characteristic finding is a well-marked aplasia of the bone-marrow. The erythroplastic tissue of the marrow is replaced by fat, in which no cellular elements are present. In regard to the pathogenicity of this condition I came to the conclusion at that time, as a result of careful review of literature and a study of the case that I was reporting, that aplastic anemia is an anemia secondary to primary marrow changes alone and is not the result of hemolysis. It is a type of anemia in which the blood changes are dependent entirely upon changes in the marrow, which, in turn, probably becomes pathologic as a result of some toxin which has a predilection for the marrow (*e. g.*, benzol). The condition is quite different from the so-called pernicious type of anemia, in which the marrow is red and hyperplastic and is presumably putting out a large number of cells which are rapidly destroyed.

**Comments.**—The present case is somewhat above the average age for patients with this type of disease. She had, as I stated before, been perfectly well up until a short time before admission, when her symptoms compelled her to enter the ward. Her symptoms were not characteristic of any severe anemia. There is no apparent cause for her condition that we can determine by the history, and none which we can find on physical examination. It is true that several times she had a rather marked eosinophilia, but repeated examination of the stools



showed no ova or parasite even after active purgation. Her anemia was intense when she first entered the hospital, and it is perfectly surprising how a person with such an anemia could be up and around. She was transfused shortly after her admission to the ward, and the reaction from this was fairly good, so that her red cells were increased rather markedly. In view of this there could not have been a complete aplasia in the marrow at that time, because the beneficial effects of the transfusion persisted for nearly two months, showing that the good results from the transfusion were not only due to the new cells which were thrown into the circulation but also stimulative effect of this transfusion upon the marrow. Three months later she was transfused again, had a well-marked rise in her count shortly after the infusion, and following this a gradual falling of the number of red cells until they had reached figures below a million. We were anxious to transfuse her before this stage had been reached, but, unfortunately, had great difficulty in finding the proper donor. She was transfused again in the early part of November, and the count rose about 500,000, returning to its previous figure within a month. The last two transfusions probably represent merely an increase in the red cells which was due to the functioning and persistence of living transfused red cells. The first transfusion represents the stimulating effect upon the marrow, while the last apparently had no effect on the marrow.



## CLINIC OF DR. JOSEPH SAILER

UNIVERSITY HOSPITAL

---

### MUMPS

MUMPS is a disease of such mild virulence and unimportant sequelæ that it has not, as a rule, excited much interest on the part of the medical profession. This is particularly true because it is self-limited and no treatment hitherto devised has been of any value whatever. Each case runs a definite course and terminates in recovery. It is usually acquired in childhood and confers practically a permanent immunity. It is only when large numbers of individuals, previously not exposed, are collected together that it becomes at all an interesting condition. Such conditions arise where large numbers of men previously living in sparsely settled rural districts are brought together for any purpose, as, for example, in industrial operations and in armies. The late war provided such conditions in the concentration camps. Camp Wheeler was a National Guard Camp, and the men first sent there were from the states of Alabama, Georgia, and Florida. The majority of these men came from rural districts, often from conditions of living that were deplorable. Many of them had no schooling whatever and the degree of illiteracy was high. They had not been thrown in close contact with others, and therefore had escaped many of the minor infections of childhood. As a result there were two extensive epidemics of measles and mumps. The former occurred chiefly in the months of September, October, and November. Altogether there were 4000 cases of measles and, as in other camps, many of these cases subsequently developed pneumonia because it was not understood at this time how to handle these epidemics. The after-care was at first inadequate because it was considered a comparatively

mild disease. The epidemic of mumps occurred later. Although there had been a few cases from time to time, it actually commenced on November 20th, and it then continued until about February 1st.

As soon as it was perceived that the epidemic was likely to be extensive a portion of the hospital was set apart for the treatment of these cases under the general supervision of Captain Hathcock. The energy and enthusiasm that he displayed in the study of these cases was remarkable. It is not, as a rule, interesting to watch large numbers of mumps, but by taking particular pains to specify the involvement of the different salivary glands, to record the frequency of complications and their character, he accumulated a mass of material, some of which he incorporated in an article on this subject. Subsequently Lieutenant Radin classified this material and published it in the Archives of Internal Medicine for September, 1918. This, I believe, is the most extensive and, I am quite sure, it is the most accurate statistical study ever made of infectious parotiditis.

There were several problems that we set ourselves to resolve:

- (1) The frequency of the involvement of the submaxillary and sublingual glands. This we found to occur much more frequently than is commonly supposed, and in a few cases one or the other of these glands was alone involved and the parotids escaped.
- (2) The frequency and nature of the complications. It is believed that all of these except cerebral mumps occurred at Camp Wheeler, and, in addition, one that had not hitherto been described. A case of cerebral mumps was observed at the hospital center in Vichy and completed my experience in the complications.
- (3) The possibility of preventing the complication of orchitis. Two methods were employed which will later be described, and both proved entirely valueless.
- (4) An effort was made to obtain cultures. In this our success was doubtful. Some anaërobic growths occurred, but they did not survive the first generation and no studies of them could be made.

There was no serious attempt made at treatment. The various drugs that were employed from time to time proved

entirely valueless and the disease practically was allowed to run its course.

A few statistics only will be given. These have all been published in Lieutenant Radin's paper, and for details reference may be made to it. Altogether there were 5736 cases treated in the Base Hospital. It was said that a few cases were treated in the Regimental Dispensaries, and although I made every effort to find out how many, nearly all the Regimental Surgeons failed to report, and the number was probably very small. This morbidity for mumps represented about one-third of all the soldiers in camp during the period of the epidemic. Each case was kept in the hospital for a minimum of three weeks. Some were kept longer on account of the occurrence of complications, but altogether at least 17,268 weeks or 332 years of service were lost to the United States Government. If the records were available and all the time could be calculated I think the figure would be nearer 350 years. This represented a serious loss, particularly as the men were being intensively trained, and every day of this training that was lost was considered important. Captain Hathcock estimated that the cost of the epidemic was at least \$1,000,000. This may be regarded as a very conservative figure.

The only advantage that may be assumed, therefore, occurred in the immunization of these men against mumps, and the fact that they had it on this side of the Atlantic rather than on the other, where the hospital facilities were, for a time at least, not nearly so adequate and where an epidemic of this nature would have proved a more serious handicap. It may easily be argued that it is of advantage to the community to permit children to become infected with mumps at a time in their life when the necessary suspension of activity for a few weeks is of relatively little importance. At least this might be done until we know more of the nature of the virus and the methods of its transmission, or until some specific and prompt therapeutic measure is devised.

**Involvement of Glands.**—In regard to the involvement of the different glands concerning which very careful observations

were made, although absolute accuracy cannot be assumed, the following figures were obtained: Both parotid glands only were involved in 2747 cases, the right parotid alone in 554, the left alone in 627. The difference is too slight to assume that the left parotid is more subject to the virus of mumps than the right. Both submaxillary glands only were involved in 16 cases, the right alone in 7, and the left in 11. There was 1 case in which both sublinguals only were involved, and 1 in which only the left sublingual was involved. Altogether, therefore, in 3928 cases the parotids only were involved; in 34 cases only the submaxillary glands; and in 2 cases only the sublingual glands. The chances, therefore, of overlooking a case of mumps because the parotids are not involved is less than 1 per cent. Complications were relatively more frequent. In 315 cases the parotids and either the submaxillary or sublingual glands were involved. In 10 cases all six glands were swollen. In 2 cases the sublingual and submaxillary glands were involved and the parotids escaped. Thus in about 6 per cent. of all cases of mumps either the submaxillary or sublingual glands are alone or together with the parotids the seat of the disease.

**Symptoms.**—The symptomatology in these cases was not different from that ordinarily observed. The first symptom is almost invariably a sense of stiffness in the jaws and the sudden distaste for sour or highly seasoned food. The latter is sometimes masked by a temporary loss of taste. Frequently the patient complained of dry mouth and drank large quantities of water to overcome it. Dr. Hathcock discovered a sign which seems to be quite characteristic and was present in all the cases examined after its recognition. This consisted of running the finger from the chin around the ramus of the inferior maxilla. There is distinct evidence of tenderness just before the angle of the jaw is reached. The patient gives a slight grimace and usually says that it hurts. This we named *Hathcock's sign*, and, as it is present before there is any apparent swelling, it seems to be of considerable diagnostic importance. I did not

have an opportunity of testing it in children, but I think there is no doubt it will be present in them also.

**Physical Signs.**—Of the physical signs, there are two. The swelling of the parotid glands occupying the outer side of the jaw bone just below the malar bone. It gives a distinct sense of elasticity. The degree of resistance gradually increases as the swelling increases. The tenderness varies considerably, but is always present and is sometimes severe. The two glands are rarely simultaneously involved, but one usually begins before the other. There seems to be no predilection for one side to develop before the other one does.

Swelling of the submaxillary gland is recognized by the same type of elastic tumor occurring just to the inner side of the lower jaw bone in front of the angle on one or both sides. This swelling may sometimes be quite large.

Swelling of the sublingual glands is recognized by small nodules occurring on either or both sides of the lower jaw just behind the chin. These are usually not large enough to give any sense of elasticity, but feel like large soft lymph-glands. If the cheek is held back by a tongue depressor the orifice of Stenson's duct is easily recognized. It appears as a slightly swollen clear papillæ with a pink areola around it. A flash-light is the best method of illumination. It is then seen on the cheek side very near the upper groove of the reflection of the mucous membrane and opposite the second molar. It is very easily recognized.

**Course.**—The course of the disease is quite characteristic. The fever is rarely high, although occasionally for one or two days temperatures exceeding 105° F. were observed. The usual duration is about five days, but in those cases in which the glands were involved successively it sometimes lasted much longer. There is usually anorexia. The patients during the first four or five days were always lethargic, resting quietly in bed and rarely making much complaint. This, however, was so characteristic of our soldiers in all forms of disease that it can hardly be regarded as a peculiarity of mumps. During the height of the swelling of the parotid gland the patient complains

of a full feeling in the ears and the hearing is distinctly less acute. There are no characteristic changes in the urine. Elaborate urinary tests were not made. It would have been impossible for the small laboratory force to have undertaken these because of the enormous amount of work that was necessary in the case of the other infections, particularly pneumonia, meningitis, and various forms of streptococcic infection.

A few of the patients had signs of general infection, that is, pains in the various parts of the body, headache, bones, and testicles. Testicular pain even when orchitis did not subsequently develop was not particularly uncommon. It probably means, what we were subsequently lead to believe, that the testicles are nearly as good a culture ground for the organism of mumps as the salivary glands, and that frequently the organism lodges there without, however, invariably producing an infection of mumps. If this surmise is correct it follows that the infection of mumps, however it is borne into the body, is blood borne to the definite seats of infection. It is to be regretted that in the early stages we did not undertake anaërobic blood cultures. They were not done partly for the reasons that have already been given. There is usually fever and, as a rule, it is low. A few cases of temporary hyperpyrexia occurred, and almost invariably when orchitis developed the temperature rose sharply. This was indicated by a secondary rise in temperature, sometimes fairly high.

Very careful studies of the blood were made. They revealed, unfortunately, nothing of importance except that in orchitis there was a mild leukocytosis between 10 and 11,000. In ordinary cases the leukocytes ranged from about 5000 to 8000, which cannot be regarded as abnormal. There was a slight preponderance of lymphocytes in the majority of uncomplicated cases. The leukocytosis of orchitis is apparently due almost exclusively to an increase in polymorphonuclears. There was, however, no change sufficiently characteristic to be of diagnostic value.

**Complications.**—It is difficult to differentiate the complications from the normal manifestations of mumps. Among those which may be regarded in the latter class are:

1. *Orchitis and Epididymitis*.—This occurred in 611 of our cases. Unfortunately, an accurate differentiation between the two conditions was not made. I was impressed with the fact that the testicles were usually exquisitely tender. Often they were more tender than they are in ordinary gonorrheal infection, and in many of the cases the epididymis was not tender, but I did not keep accurate figures, and there were cases in which the reverse was true. If the mumps were unilateral the orchitis was usually unilateral and on the same side, although exceptions to this were also noted. Whether this is of any real significance I am unable to say, but it was at least an interesting observation.

There seemed to be no definite time at which the orchitis developed, and in a few cases it occurred before there was definite involvement of the salivary glands. The first signs of this complication are pain in the testicles and epididymis. This is quite severe. In the course of one or two days the testicles begin to swell, and then the pain seems to grow less, although it does not entirely subside. The swelling may last for a considerable period of time. About ten days was the maximum, but usually in less time than this period the swelling disappeared and with it the pain disappeared also.

Minor complications of orchitis were four: First, backache, which was most frequent and occurred early. Second, nausea and vomiting. This was also fairly common on the first day of the orchitic involvement, but very soon subsided. Third, retention of urine. This was not particularly common, but was frequent enough to make us believe that it had a definite relation to the orchitis. Fourth, nocturnal pollutions. It was not always possible to find out how frequently they occurred, but at any rate they did occur in a number of cases. Whether they were more frequent among the men suffering from mumps than among the soldiers in the camp at large was impossible to determine. It is my personal impression that they were not.

2. *Pancreatitis*.—There has been much dispute as to whether the pancreas is ever involved or not. It was not possible to determine whether it was swollen or not even in thin individuals. The symptomatology upon which the diagnosis was based con-



sisted of pain and tenderness above the umbilicus and in the median line. This tenderness was diffuse and it was not possible to determine that deep inspiration caused the pancreas to slip down from the palpating finger and the tenderness to disappear. This sign, however, I have observed in other forms of pancreatitis. Second, loss of appetite and nausea. These are quite characteristic and there is occasionally some vomiting. Third, the appearance of jaundice. If this were common it might be regarded as fairly decisive, but as a matter of fact we noted it in only 4 cases. Consequently, it cannot be regarded as a certain indication of involvement of the pancreas. Fourth, the manifest increase in the general symptoms. The patient is prostrated. The pulse becomes more frequent and of lessened volume. Just when the pancreatitis, if it is pancreatitis, occurs it is difficult to say. Many of the patients had gastro-intestinal discomfort in the beginning. In others it did not develop until the disease was well advanced. Lieutenant Radin reached the conclusion from the figures that it was most common from the fifth to the eighth day of the disease. Altogether, typical symptoms of involvement of the pancreas were comparatively rare. There were only 14 cases in which we felt reasonably certain.

3. *Involvement of the Thymic Gland.*—The evidence on which this diagnosis was made was somewhat inadequate, but if the diagnosis is unwarranted the symptoms are so extraordinary and characteristic that we must assume a very definite lesion for their causation. The first thing noticed in all our cases was a distinct swelling over the manubrium. This was a localized cutaneous edema, pitting slightly on pressure. It extended from about the cricoid cartilage as low as the middle of the gladiolus and on either side to about the midclavicular line. The area of swelling was circular or oval in shape. It was not sharply delimited. There was no localized tenderness and no redness. In all of the 6 cases the thymic sign was quite easily elicited, that is to say, if percussion was made over the manubrium with the head bent forward and then in the same spot with the head bent backward, the dull note in the former position changed to a fairly loud resonant note in the latter. This is supposed to be



due to the fact that the thymus is pulled upward by the thyrothymic ligament, but the value of this sign has been denied. Major Wheat kindly undertook to make x-rays and convinced himself and us that there was an abnormal shadow just back of the manubrium. This might represent an enlarged thymus. At any rate we reached the conclusion that the peculiar syndrome described was possibly the result of a swelling of the thymus and that the edema was due to the obstruction of the lymphatic channels supplying the superadjacent skin. It is, of course, an extremely rare complication, having been seen in only 6 cases, or about  $\frac{1}{16}$  of 1 per cent.

4. *Cerebral Mumps*.—No cases of this were seen at Camp Wheeler, but during a much smaller epidemic that occurred at the Hospital Center at Vichy 1 case occurred in the Hospital for Contagious Disease at that place. Careful statistics were kept of all the contagious cases, but unfortunately I have not access to them now. There were, however, about 100 cases of mumps during the period from August, 1918, to January, 1919, at which time I was ordered home. The patient, an ordinary parotid type of mumps without orchitis involvement, one day suddenly became distinctly maniacal, tried to jump from the window, the ward was in the third story of the hotel, was excited, the face was flushed, and the pupils slightly dilated. A spinal puncture was made, but no increase in pressure was found. I had at this time recently read Trusseau's description, quoted by Dieulafoy, of this condition, which corresponded so closely to the case under observation that I ventured to make a very favorable prognosis, and this was entirely justified by the subsequent course of the case. The following day the patient was perfectly rational and proceeded to an uninterrupted and uncomplicated convalescence a few days later.

It may be interesting at this point to quote the report of one of Trusseau's 2 cases. He was seen in 1853 and was seventeen years of age. "Patient was seized with a high fever, very frequent pulse, delirium, cough, vomiting, serous and involuntary diarrhea." Various diagnoses were made. "The following morning the state of the invalid had not changed, but perhaps

it was not quite so bad. We were told of a slight accident which had been perceptible during the night. The scrotum was swollen and one of the testicles painful. This was the single organic lesion that could be noted, and surely it was hardly of a nature to account for the symptoms of which we were witness." Trusseau recalled a similar case and made a favorable prognosis, and in a short time the patient recovered. The condition in our case was apparently less severe than in either of the cases described by Trusseau, but it belongs undoubtedly to the same category.

Various other complications arose, but they can hardly be ascribed to mumps. Thus there were 20 cases of otitis media. This was less than the average of all cases in the hospital, where, for a time, until measures were taken to check it, otitis media was exceedingly common. There were 3 cases of mastoiditis. Numerous cases of this condition also occurred among the other patients. There were 1 or 2 cases of urethral discharge of a temporary character and not gonorrheal. One might conclude that it was less common among our patients than in the camp at large; 9 cases of bronchitis were noted. As coughs were exceedingly common, these may be regarded as purely accidental, as were also 6 cases of pneumonia. Indeed, regarding the general incidence of pneumonia in the camp, it almost seemed as if the mumps had some effect in preventing the occurrence of this condition. I had feared an increase of pneumonia as a result of mumps, but my fears not only were groundless, but were contrary to what actually occurred. In one man and in one woman the breasts were tender. In both cases the tenderness was very slight. There was no apparent swelling, and we could only conclude that this was also an accident and probably in no way associated with the mumps.

**Prognosis.**—The prognosis was good throughout. There were no deaths. We were not able to follow the cases in order to determine whether any testicular atrophy occurred. We heard of no instances from the regimental surgeons. Presumably it did occur in some cases and it would have been interesting if we could have obtained some definite statistics.

**Treatment.**—The treatment was most unsatisfactory from the standpoint of definite result. Rest in bed, mild catharsis, light diet, the application of heat and mild counterirritants to the salivary glands, the application of cold to the testicles, and various methods of supporting the testicles when they were swollen seemed to give doubtful relief. Gargles were also employed, particularly a weak Dobell's solution, but I cannot say that I observed any particular benefit. Practically all the patients received instillations into the nose of 2 drops of some antiseptic in oil twice a day. This was done for the reason that otitis media had become such a serious epidemic in the hospital that Captain Rau and I decided some active measure should be taken for its prevention. We hit upon the method of the instillation of some antiseptic in oil into the nostrils of all patients in the hospital, and the mumps patients were treated as were the rest. The usual solutions were 2 per cent. phenol in oil and 3 per cent. tincture of iodine in oil. Whether as a result of this measure or because the epidemic of otitis media was near its end, I do not know, but Dr. Rau and I were strongly of the conviction that our measures had been effective because the otitis media almost immediately stopped and thereafter was not a serious complication in any of our cases. Whether this treatment is responsible for the very small number of cases of otitis media in our 5000 mumps patients I do not know. I should be strongly inclined to use it again if I should ever meet with similar conditions, that is, a greatly overcrowded hospital with an enormous preponderance of respiratory diseases.

We tried two methods to prevent or lessen the incidence of orchitis. Lieutenant Lewis was strongly of the opinion that the infection of the testicles took place through the urethra. He therefore insisted on vigorous prophylactic measures to prevent the patients from carrying the infection on their hands. These consisted of careful washing of hands before micturition, the application of mild antiseptics to the meatus, and the wearing of linen bags. These measures were absolutely ineffective. The percentage of orchitis among the men so treated was actually slightly higher than in the others.

At my suggestion Captain Hathcock put every other patient upon 5 grains of hexamethylamin four times a day. This was continued in these cases throughout the course of the disease. The difference in the incidence of orchitis in the two groups, of 200 cases, was 37 in one and 38 in the other in favor of those who had been treated. This indicates that the hexamethylamin was of no value in preventing the occurrence of orchitis. It is only another evidence in support of the fact that the infection was carried to the testicles by the blood.

Salicylic acid seemed to relieve the pain and to diminish the fever. It was practically the only drug employed.

The social question in this epidemic seemed to me to be the most important. It has been said that there should be no usual diseases of childhood. It has been argued that all infections must leave some trace deleterious to the individual. It is probably true that, if these men had been left to the blighting effects of isolated farm life that were manifested in the body as well as in the mind, because I have rarely seen such a group of thin, shambling men as were the members of the 31st Division when they first came to camp, many of them might have escaped mumps throughout their life.

On the other hand, it was clearly demonstrated by our camp life that when they are brought together, mingle with their fellows, and have the intellectual and physical stimulation that this mingling involves, they are almost sure to acquire these infections in early adult life, and at the present time we have, unfortunately, no known means of preventing contagion either of mumps, measles, chicken-pox, or scarlet fever. They had the disease certainly as severely as they would have had it in childhood, and it was our impression that on the whole it was more severe than those cases we had seen, practically always in children, before coming to the camp.

It would seem, therefore, that for them nothing had been gained by escaping the early infection which comes to almost all children in urban or thickly settled farming communities. It may also be argued that the loss of time to an adult is considerably more important than it is to a young child. Therefore it

is not impossible that at the present time it is of advantage to the population as a whole to have these practically inevitable diseases early in life and secure the permanent immunity that each of them entails. It is true that a few of our patients gave definite histories of having had mumps previously, and one patient insisted that he had had them twice. It is, of course, possible that these histories were inaccurate, and Captain Hathcock, who was a southern practitioner and who had a large experience, insisted that they were not correct, but it is also notable that the nurses, corps men, and physicians were thrown in daily contact with these mumps cases and were not infected. This may have been due to their wearing masks, but I regret to say that it was very difficult to enforce this rule absolutely, as constantly I was forced to call the attention not only of the corps men and nurses but also of the physicians to the fact that their masks were worn wholly below the chin. Practically all of these were from urban settlements and insisted that they had already had mumps and were in no danger. The outcome seemed to bear out this assertion. Altogether only 29 cases developed among the Medical Department, and some of these were in the Ambulance Company and in men not more exposed than were the men in the camp.

It is therefore my impression, which I offer with the utmost diffidence, that it is of little use to adopt any vigorous prophylactic measures to prevent the occurrence of mumps. Surely the three weeks' isolation that we employed in the hospital was of little if any value. It might be added that the general physical and mental improvement in all of these men during their stay in camp was most remarkable, and that the advantages of the wider social life far outweigh the dangers of the infantile diseases. A finer group of soldiers than these men when they were ordered abroad it would have been difficult to find in any other camp.



## CLINIC OF DR. EDWARD H. GOODMAN

UNIVERSITY HOSPITAL

---

### SIGNIFICANCE OF HEART MURMURS IN YOUNG INDIVIDUALS

THERE is at the present time a growing tendency to regard murmurs in a somewhat different light than was the case a decade or two ago. Then it was the custom, after discovering the murmur, to interpret it on the basis of valve lesion, and it followed that the next step was the diagnosis. Further than that few teachers brought their students, leaving them with the impression that murmurs were the Golden Fleece of the physical examination, that finding them was the achievement of an important quest, and that having found a murmur they might now regard themselves as having taken their first step toward clinical immortality. Today we are in a position to offer another teaching, a truer one, but a difficult one to find acceptance with our students of a previous decade, but which with less difficulty is appreciated by the student of today, namely, that murmurs when compared with all there is to be learned from an examination of the heart are so significantly insignificant that auscultation has been held to be the least important of the methods of physical examination of the heart.

It will be my endeavor to present for your consideration the significance of murmurs heard over the chest, which are definitely associated as far as auscultation and reasoning permit us to judge, with the heart, for some murmurs are clearly cardiac, some are as clearly extracardiac, and some are not so clearly differentiated. Furthermore, some murmurs, in the presence of other signs, can be interpreted as indicating diseased valves,

and some murmurs, with exactly the same quality but without certain other signs, must be regarded as meaning no valve defect, and hence of little significance. It will be apparent that, unless we are to save ourselves hopeless confusion, some sort of classification becomes immediately necessary, so that we can proceed to give consideration in a consecutive way to the significance of murmurs, "to assess the value of a murmur" as Mackenzie says. So that the difficulties attending this bold intention and procedure may be fully appreciated a passage from Mackenzie's book, *Principles of Diagnosis and Treatment in Heart Affections*, is quoted, as showing how this eminent scholar regards such intrepidity: "At times physicians of experience will admit that certain murmurs may not have a serious significance; in saying this many of them appear to imagine they have made an important contribution to the subject. But having failed to point out at the same time how a murmur may be valued so as to discover when it is of significance and when it is not, they have really failed to carry forward our knowledge in any respect. The result of this imperfect comprehension of its meaning is that, at present, every graduate leaves the hospital with but the vaguest notion of how to assess the value of a murmur. In other words, teachers and writers have hitherto shrunk from laying down clear and definite lines for guidance, and I know that in the attempt I am about to make to do this I undertake a very hazardous enterprise. I do so, however, not with the idea that I have finally settled the matter, but in hope that others who follow me may test my observation, and so extend the knowledge of this very important subject."

Although a number of schemes of classification might be offered, and any one prove to be a good working plan, the one that is the basis of the following discussion is one which closely follows that of Barker:

I. *Intracardial Murmurs:*

1. Organic (due to diseased valves): A. Systolic. B. Diastolic.

2. Non-organic (not due to diseased valves): A. Systolic. B. Rarely, very rarely, diastolic.



## II. *Extracardial Murmurs:*

1. Pericardial (friction sounds): Pericarditis.
2. Pleuropericardial (friction sounds): Pericarditis-pleuritis.
3. Pleural (splashing): Hydropneumothorax.
4. Pulmonary: Cavity.
5. Gastric: Distended stomach—air and liquid.
6. Aortic: Atheroma-aneurysm.

As the title of this clinic states, attention will be devoted to intracardial murmurs alone, since those for whom this contribution is intended will find no difficulty in distinguishing these murmurs from those arising outside the heart.

## I. INTRACARDIAL MURMURS

1. **Organic Murmurs Due to Diseased Valves.**—A. **Systolic.**  
—(a) *Apical Systolic.*—Granting that there is a systolic murmur over a certain valve area, how is one to tell whether it is due to a diseased valve, having regard now only to the murmur? Is there a peculiar quality to the murmur? We are told that systolic murmurs at the base, when due to valve defect, indicate narrowing, obstruction, or stenosis, and that the quality of a stenotic murmur is rough and churning; furthermore, at the mitral or tricuspid area systolic murmurs caused by a diseased valve indicate leakage, regurgitation, or insufficiency, and the quality of a regurgitant murmur is musical or perhaps rough. These statements are, no doubt, true, but it is equally true that organic murmurs may be soft and blowing, resembling the so-called functional and physiologic murmurs, and there will arise many occasions when it will be impossible to decide from the murmur alone whether one is dealing with a diseased or healthy valve. Therefore, are we justified in reading into a murmur an interpretation other than that here is an adventitious sound whose nature or method of production is uncertain? Decidedly not, and if the murmur does not tell us what we are seeking, then of what use is it? It has this object, and a most important purpose it is, that, given an anamnesis pointing to some cardiac disease with an antecedent history of rheumatic fever and an examination revealing the presence of

heart trouble, the murmur enables us to render the diagnosis accurately or more nearly so. No one would presume to diagnose mitral insufficiency when no murmur was heard, despite the history of rheumatic fever, of pain and breathlessness, the finding of hypertrophy, tachycardia, and poor response to exercise; neither should he diagnose mitral insufficiency in the presence of a murmur when all the features named above are absent. No one would hesitate about the treatment of these two hypothetical cases—the first demands attention; the second, no therapeutics. To himself no physician would for a moment give other than unfavorable prognosis to the first, and should not, to himself and to the patient, give other than the happiest encouragement and reassurance.

It resolves itself then to this fact, the incontrovertible fact, the scarcely debatable fact, that systolic murmurs should be used for diagnosis and for nothing else, but it must be insisted upon that the diagnosis of a valve defect is not made in the murmur alone, but on other signs as well. We shall see later that there are some systolic murmurs which are not due to diseased valves, and it were a simple matter, indeed, to differentiate these if there were anything peculiar about the quality of the murmur. However, granting that in patient A an apical systolic murmur has been found and correctly interpreted to mean mitral insufficiency, while in patient B a similar murmur has been detected and correctly regarded as a physiologic or innocent murmur; the diagnosis, presumably correct in both instances, being given the patients, the next thing they will wish to know is what bearing these abnormalities will have upon their future; how many years they may expect to live; in short, what is the prognosis? Suppose A is a bank clerk and B is an athlete. A is satisfied to be restricted in his exertions and B is anxious to indulge in sport; you tell B he may, and because of the physiologic murmur you expect no harm to result. In course of time both return to the consulting room, A in perfect condition, B with pain, breathlessness, cyanosis, palpitation, dizziness, and exhaustion on exertion, with displaced apex-beat to the left, hypertrophy, tachycardia, and distention of the veins (all of which phenomena which for

the sake of brevity in the course of this clinic will be referred to as "the myocardial syndrome"), but with the same murmur—what is the verdict now? Did B have a physiologic murmur or was it mitral insufficiency? Can he still indulge unrestrainedly in athletic sports? The murmur has not changed, but you change your opinion, and the opinion is changed because of things far more important than murmurs.

Lewis when asked if he attached significance to such apical murmurs when the heart is enlarged, answered that he attached significance to the *hypertrophy*. Army experience has taught us to disregard apical murmurs when there is no history of rheumatic fever, when there is no enlargement, and when the response to exercise is good, and Lewis' dictum must be recalled, "Systolic murmurs are to be neglected in arriving at a prognosis in young men." Regarding the murmurs heard in the course of acute fevers the same holds good, namely, that if the "myocardial syndrome" is absent, and if there is a well-marked sinus arrhythmia, the murmur is of no significance.

(b) *Pulmonary Systolic*.—Systolic murmurs heard over the second, third, or fourth costal cartilages (left) are of frequent occurrence, and when heard, thrills should be sought for. The two lesions giving rise to a murmur and a thrill are pulmonary stenosis and patent ductus arteriosus. The former is a rare condition and a serious one, and can be diagnosed only when there is a thrill as well as the murmur and undoubted evidence of cyanosis. A patent ductus presents variable signs of which a murmur is constant and a thrill usually so, but the condition is not a serious one provided there is no cyanosis, hypertrophy, and if the response to exercise is normal. In the absence of thrills and no evidence of the "myocardial syndrome" a systolic murmur at the pulmonary area is of no significance.

(c) *Aortic Systolic Murmurs*.—This murmur is less frequently heard than the pulmonary systolic and does not necessarily mean stenosis. Aortic stenosis in the absence of insufficiency is a rare condition, but if regurgitation is present, then a systolic thrill with an anacrotic pulse would lead one to suspect the condition. We shall learn later that aortic insufficiency is of itself

heart trouble, the murmur enables us to render the diagnosis accurately or more nearly so. No one would presume to diagnose mitral insufficiency when no murmur was heard, despite the history of rheumatic fever, of pain and breathlessness, the finding of hypertrophy, tachycardia, and poor response to exercise; neither should he diagnose mitral insufficiency in the presence of a murmur when all the features named above are absent. No one would hesitate about the treatment of these two hypothetical cases—the first demands attention; the second, no therapeutics. To himself no physician would for a moment give other than unfavorable prognosis to the first, and should not, to himself and to the patient, give other than the happiest encouragement and reassurance.

It resolves itself then to this fact, the incontrovertible fact, the scarcely debatable fact, that systolic murmurs should be used for diagnosis and for nothing else, but it must be insisted upon that the diagnosis of a valve defect is not made in the murmur alone, but on other signs as well. We shall see later that there are some systolic murmurs which are not due to diseased valves, and it were a simple matter, indeed, to differentiate these if there were anything peculiar about the quality of the murmur. However, granting that in patient A an apical systolic murmur has been found and correctly interpreted to mean mitral insufficiency, while in patient B a similar murmur has been detected and correctly regarded as a physiologic or innocent murmur; the diagnosis, presumably correct in both instances, being given the patients, the next thing they will wish to know is what bearing these abnormalities will have upon their future; how many years they may expect to live; in short, what is the prognosis? Suppose A is a bank clerk and B is an athlete. A is satisfied to be restricted in his exertions and B is anxious to indulge in sport; you tell B he may, and because of the physiologic murmur you expect no harm to result. In course of time both return to the consulting room, A in perfect condition, B with pain, breathlessness, cyanosis, palpitation, dizziness, and exhaustion on exertion, with displaced apex-beat to the left, hypertrophy, tachycardia, and distention of the veins (all of which phenomena which for

the sake of brevity in the course of this clinic will be referred to as "the myocardial syndrome"), but with the same murmur—what is the verdict now? Did B have a physiologic murmur or was it mitral insufficiency? Can he still indulge unrestrainedly in athletic sports? The murmur has not changed, but you change your opinion, and the opinion is changed because of things far more important than murmurs.

Lewis when asked if he attached significance to such apical murmurs when the heart is enlarged, answered that he attached significance to the *hypertrophy*. Army experience has taught us to disregard apical murmurs when there is no history of rheumatic fever, when there is no enlargement, and when the response to exercise is good, and Lewis' dictum must be recalled, "Systolic murmurs are to be neglected in arriving at a prognosis in young men." Regarding the murmurs heard in the course of acute fevers the same holds good, namely, that if the "myocardial syndrome" is absent, and if there is a well-marked sinus arrhythmia, the murmur is of no significance.

(b) *Pulmonary Systolic*.—Systolic murmurs heard over the second, third, or fourth costal cartilages (left) are of frequent occurrence, and when heard, thrills should be sought for. The two lesions giving rise to a murmur and a thrill are pulmonary stenosis and patent ductus arteriosus. The former is a rare condition and a serious one, and can be diagnosed only when there is a thrill as well as the murmur and undoubted evidence of cyanosis. A patent ductus presents variable signs of which a murmur is constant and a thrill usually so, but the condition is not a serious one provided there is no cyanosis, hypertrophy, and if the response to exercise is normal. In the absence of thrills and no evidence of the "myocardial syndrome" a systolic murmur at the pulmonary area is of no significance.

(c) *Aortic Systolic Murmurs*.—This murmur is less frequently heard than the pulmonary systolic and does not necessarily mean stenosis. Aortic stenosis in the absence of insufficiency is a rare condition, but if regurgitation is present, then a systolic thrill with an anacrotic pulse would lead one to suspect the condition. We shall learn later that aortic insufficiency is of itself

a serious lesion, and the addition of stenosis makes us regard it no more and no less so. In young individuals aortic systolic murmurs rarely indicate lesions of the aorta itself.

(d) *Tricuspid Systolic Murmurs*.—A murmur heard over the tricuspid area is rarely heard in health, is frequently heard in hearts with other valvular lesions, and is not infrequently heard in patients presenting the "effort syndrome" (Lewis). I have never heard it in healthy individuals at rest, but once in acute valvulitis affecting the tricuspid valve, and not often after exercise in healthy individuals, although Lewis believes it is not uncommon in the last named. Prognostically, the murmur has no significance.

B. *Diastolic Murmurs*.—(a) *At Apex*.—Diastolic murmurs wherever heard indicate damage, and although this statement may be questioned, it is nevertheless true that diastolic murmurs are rarely if ever heard in healthy hearts. Therefore, having heard a diastolic murmur, it rests with the physician to label it on the basis of pathology without the confusion of the probability of it being a physiologic condition. With diastolic murmurs the axiom holds as it does with systolic murmurs, that their main revelation is diagnostic.

A diastolic murmur at the apex, whether it occurs early in diastole, or in middiastole, or in late diastole (presystolic), in the absence of signs pointing to aortic insufficiency, indicates mitral stenosis. Of all valvular defects, this is the most interesting, for the reasons that oftentimes there is no murmur, the murmur is variable in its exhibitions, and in addition to being simply a peg upon which to hang a diagnosis, the murmur has a prognostic significance as well—a virtue, if it may be so called, shown by no other valve lesion. When there is no murmur it is still possible to recognize an abnormality of cardiac mechanism, which should arrest attention so that efforts may be made to bring out the murmur.

Auscultation, in a case of uncomplicated mitral stenosis, reveals in the vast majority of cases a first sound so manifestly not the first sound heard in health that its features should be closely studied. It is a first sound that is heard in no other

valve lesion so characteristically—it has a clear, ringing, snappy sound that defies description, but which may, in a measure, be imitated by grasping the slack of a piece of stout material in both hands and pulling it taut, or by tapping forcibly with a staccato blow of the finger the back of the hand, the palm being held tightly against the ear.

Of almost equal importance with the first sound rank the features recognized by palpation. The apex-beat is circumscribed and generally not displaced, striking the hand sharply and immediately receding (systolic tap or shock). It is not the apex-beat of a normal heart or of an hypertrophied organ, and is an important phenomenon when a thrill is absent. A presystolic thrill is by no means always present, and when present does not always indicate mitral stenosis, but a presystolic thrill should arrest attention when it terminates in a systolic tap or shock, and when auscultation gives us the snappy first sound and a murmur is present, the evidence is clear.

The murmur, however, of mitral stenosis is most elusive and variable—it may be present one day and absent the next; it changes its qualities from day to day, from hour to hour, and in one interesting case in the army the features changed while one of the cardiovascular board was bringing the soldier to my office, a distance that consumed no more than two minutes of time. The quality of the murmur is harsh. When the murmur is definitely late in diastole (presystolic) it becomes more loud as it terminates in the loud first sound; or the murmur may start in the middle of diastole or it may occupy the entire diastolic period.

The part of diastole occupied by the murmur has prognostic significance far greater than the diagnostic, for we are enabled by the timing of this murmur to gauge its progress. The nearer the murmur is to systole and the shorter in time it is, the more recent the lesion or the more stationary and non-progressing is the defect, and the healthier is the heart muscle. The farther the murmur extends back into diastole, becoming middiastolic or entirely diastolic, the more serious the condition. For instance, if an individual presents a short presystolic murmur on first



examination, a year later a murmur beginning in middiastole, and a year later a murmur occupying all of diastole, the prognosis should be guarded. To quote Mackenzie for another illustration: "When you find mitral stenosis in the very young, accompanied by presystolic and diastolic murmurs, you know you have a grave condition to deal with. When you find only presystolic murmur in a patient aged about forty, who has a history of rheumatic fever in childhood, then you know that the rate of stenosis is slow and that it may be stationary."

When no murmur is heard, but attention is directed to the possibility of there being mitral stenosis by reason of the palpatory and the auscultatory findings, there is a possibility of hearing the murmur if the patient is examined not only in the upright position, but on his back, abdomen, and side, and after exercising. Should this procedure be followed and no murmur be heard, it should be repeated on succeeding days, until no shadow of doubt remains as to the true state of affairs. These remarks apply to cases of stenosis without insufficiency, for when a systolic murmur of a regurgitation appears the quality of the first sound is modified. However, by studying closely the insufficiency, stenosis if present will not be overlooked. The murmur accompanying aortic insufficiency and known as a Flint murmur will be properly recognized by the associated features of the aortic valve defect.

(b) *At Base*.—A diastolic murmur heard in the second right intercostal space or many times over the left side of the chest, transmitted along the sternum, with absence of the second sound, displacement of the apex-beat to the left, heaving feel of apex-beat (*choc en dôme*), hypertrophy of left ventricle, vascular signs (marked pulsation of vessels, Corrigan pulse, capillary pulse, systolic tone in arteries, brought out in brachials by holding arm above head), with certain blood-pressure findings (marked discrepancy between leg and arm pressures, increase in pulse pressure in arm and leg, particularly in the latter)—all these make the diagnosis of aortic insufficiency a certainty. The diastolic murmur heard in the third left intercostal space in cases of mitral stenosis should cause no confusion (Graham Steele



murmur), as all signs of hypertrophy are absent, as well as the vascular and blood-pressure phenomena mentioned above.

**2. Non-organic Murmurs Not Due to Diseased Valves.—**

**A. Systolic.**—Systolic murmurs are heard with great frequency either at base or apex and may or may not indicate valve, muscle, or vascular disease. It is my opinion that many practitioners today are stigmatizing individuals possessing such murmurs with the diagnosis of valvular heart disease and are thereby condemning them to a life of semi-invalidism regardless of certain features other than the murmur. These murmurs are variously classed as innocent, accidental, physiologic, or functional. The terms "innocent," "accidental," and "physiologic" are synonymous, and should be used to describe those murmurs heard in a perfectly normal heart. We know these hearts are normal because the individuals lead strenuous lives without heart failure, because at death there is no demonstrable lesion, and because there is no increase in the size of the heart and the tolerance to exercise is good. The term "functional murmur" is used to include all murmurs not due to organic valve disease (cardiac dilatation), and such murmurs are accompanied by increase in size of the heart and by poor exercise tolerance.

The innocent murmurs have been called in the army "apex systolic" and "pulmonary systolic," but there is a much more common murmur which must be recognized and properly pigeon-holed. I speak of the cardiorespiratory murmur.

(a) *Cardiorespiratory Murmur.*—In our examinations of about 60,000 men at Camp Jackson these murmurs were heard so frequently that we found it impossible to make records of all, but all were accepted for full military duty. The murmur is generally systolic in time, heard for the most part at the apex, although not infrequently at the angle of the scapula. It is heard best during deep inspiration and expiration, being loudest in the middle of both, and disappearing at the end of full inspiration and complete expiration. In reality it is a pulmonary breath sound accompanying each systole and having absolutely no significance.

(b) The *pulmonary systolic* and (c) the *aortic murmurs* have been mentioned under Organic Murmurs.

(d) *Apex systolic murmurs* may or may not indicate mitral leakage, and from the murmur alone we learn nothing. We have said the main distinction between the physiologic and functional murmur lay in two things: the size of the heart and the efficiency of the muscle (tolerance to exercise). Supposing two patients present themselves, both having murmurs, but one with the "myocardial syndrome," and the other with no hypertrophy and with good response to exercise—which is the sicker man? Supposing, again, two other patients consult you, one has a murmur, but all the other signs are absent; the other has no murmur, but he has the "myocardial syndrome"—which is the sicker man?

Are you not relying on something besides murmurs in the most important feature of heart conditions, namely, prognosis? Are you assisted at all in prognosis by a systolic murmur? What difference does it make to a workman if he has a murmur but can do the hardest work; but what a great difference if he has no murmur and is unable to walk up a flight of stairs with comfort? "What is essential to us as physicians is to recognize that a systolic murmur, whether variable or persistent, may be of no significance as far as the future of the patient is concerned, and that when it is the only abnormal sign present, and the response to effort is good, it implies neither cardiac disease nor cardiac impairment" (Mackenzie).

Let me briefly summarize the foregoing:

Diastolic murmurs generally indicate valve disease and systolic murmurs may or may not. We know how seriously we regard mitral stenosis and aortic insufficiency simply because we have made the diagnosis from the murmur. Now is not that about all the murmur has taught us? We know these lesions are serious because the myocardium suffers most and because nearly always we see these individuals eventually decompensate. We do not recognize this eventuality by the murmur necessarily, we may recognize it by the most casual inspection. Do we vary the treatment if decompensation occurs in mitral stenosis or in aortic insufficiency? Is not the treatment based on the degree

of muscle disability regardless of the valve defect? Do we need the murmur for anything except for the diagnosis? It is interesting to label a disease no doubt, but the patient wants to know how much he can do without damage to himself, what is his margin of safety, and how much this valve lesion will shorten his days. Can you answer these questions from the diastolic murmur alone, except possibly the murmur of mitral stenosis?

With systolic murmurs the case is different. Some are physiologic, some functional, and some organic, but does it really matter whether you confuse these, and mistake the true diagnosis, provided you are able to recognize the condition and behavior of the muscle? To diagnose a serious valve lesion is to render a man a great service, for you can perhaps prevent him from overexertion, but the dangers of overexertion we have learned from muscle, not from valve. The more one studies muscle the greater will be his belief in the insignificance rather than in the significance of heart murmurs in young individuals.



## CLINIC OF DR. O. H. PERRY PEPPER

UNIVERSITY HOSPITAL

### HODGKIN'S DISEASE WITH JAUNDICE AS AN EARLY SYMPTOM<sup>1</sup>

#### Illustrative Case.

**DR. PEPPER:** To whom was this patient assigned for study?

**MR. JONES:** To me.

<sup>1</sup> Among the hours of instruction arranged for the section of the senior class on duty during their medical trimester, as clinical clerks on the Medical Service of Professor Alfred Stengel at the Hospital of the University of Pennsylvania two hours a week are devoted to what is called "new case discussion." In these hours are presented only those patients who have been in the hospital less than three days. This is done in order that the students shall see cases which have not yet been fully studied and shall have an opportunity to appreciate the difficulties of the practitioner and the consultant. Also that they shall learn the necessity of formulating a working opinion from the evidence at hand without waiting for the results of the indicated chemical, bacteriologic, or roentgenologic investigations. In this respect the patients chosen contrast sharply with those discussed in formal ward rounds and seen at clinical conferences.

As a rule, a patient who has been in the ward forty-eight hours will have had, except in an emergency, little more study than a history, a physical examination, a urinalysis, a blood count, and observations as to temperature, pulse, respiration, quantity of urine, and frequency of defecation. The student to whom the patient has been assigned will have taken the history, made a complete physical examination, and at least a preliminary urine analysis and blood count. He is expected to fill out within forty-eight hours of the assignment of the patient to him a "diagnosis blank," giving a tentative diagnosis, the evidence upon which this is based, the special examinations he would suggest to confirm or disprove the diagnosis, and lastly the preliminary treatment he would institute at this time. This may be used as the basis for discussion of the case.

In this clinic an attempt has been made conscientiously to reproduce one of the "new case discussions" which occurred this fall, and in which the diagnosis was correctly arrived at. This will serve not only as an example of such "new case discussion" but also as an accurate report of an interesting case.

DR. PEPPER: Mr. Jones will now present the patient's history and the abnormal physical findings and such laboratory work as he has performed.

MR. JONES (reading): Patient is a colored male, age twenty-two, admitted October 12, 1919.

*Chief Complaint.*—Jaundice and constipation.

*History of Present Illness.*—Six months ago he caught a heavy cold from exposure to rain and cold. About a week later he had discomfort in the "stomach" without, however, any nausea, vomiting, or severe pain. Four or five days after onset of this discomfort in the upper abdomen he discovered that his sclerae were yellow. Since that time he has been continuously jaundiced, but he thinks that the jaundice has varied at times in intensity. He also has continued to be "uneasy in the stomach," especially after eating, but of late this symptom has been less annoying. From the onset of his jaundice he has been very constipated and has had to take a bottle of magnesium citrate every night. He has noticed that his stools have been whitish in color. For about four months he has been greatly troubled by a generalized pruritus. He was in a hospital for a few days in June of this year and was told he was suffering from acute cholangitis. The treatment did him no good.

Following the onset of his trouble he lost considerable weight, perhaps as much as 40 pounds, but he states that during the past three months he has regained some weight and that he is now 20 pounds under his normal weight. He has not been eating very freely, as he has had little appetite and seemed to feel better if he did not eat very much.

Patient does not volunteer any complaint about the obvious enlargement of his neck, but on questioning states that about a month before the onset of his jaundice a friend who was rubbing his neck for "rheumatism" noticed lumps in the right side of his neck. These became quite large within a short time and extended from "near his ear to his collar bone." He is very positive that within the past three months the glands in the lower part of the mass have remained stationary in size, and that the smaller glands higher up have actually decreased in size.

The patient says he does not really feel sick, but is worried by the continued jaundice, constipation, itching, and loss of weight.

*Previous Medical History.*—Has had transitory indigestion from improper food occasionally. Gonorrhea six years ago. No chancre. A physician recently treated him for supposed lues and produced a severe salivation.

*Family History.*—Mother dead, the cause unknown. No brothers or sisters.

*Social History.*—Truck driver for four years, "handling milk with a lot of heavy lifting." He was a cook before that. Single. Lives with aunt. Food good. Always moderate eater; 4 or 5 cups of coffee a day, 2 of tea, no alcohol. Has not smoked for three months.

DR. PEPPER: In brief, Mr. Jones' patient has been deeply jaundiced for six months, and was diagnosed at the onset as being a case of acute cholangitis. The glands of his neck have been swollen for a somewhat longer period and there has been some variation from time to time in their size. The only symptoms now complained of by this patient are jaundice, intense itching, and constipation. Mr. Jones will now give us the important findings in the physical examination.

MR. JONES: The patient shows marked yellowness of the scleræ, but the jaundice cannot be seen in the skin because of the racial pigmentation. There is considerable enlargement of the anterior cervical and supraclavicular glands on both sides of the neck, but more marked on the right. The largest gland is just above the right sternoclavicular joint; this mass is about the size of a goose egg. The masses are not tender and show no sign of breaking down. The skin is not adherent to the mass. The heart and lungs are negative. The abdomen shows scratch marks. There is tenderness in right upper quadrant and the liver is doubtfully palpable. The spleen is not palpable. In other respects my examination is negative.

DR. PEPPER: Have you had time to examine the urine and blood?

MR. JONES: The urine is amber colored, cloudy, and has a

flocculent sediment. The specific gravity is 1015; the reaction alkaline. There is no albumin or sugar, but the tests for bile are strongly positive. Microscopically, nothing was found but a little epithelium. The blood count is:

Erythrocytes, 4,100,000

Leukocytes, 5,200

Hemoglobin, 72 per cent.

Differential count:

Polymorphonuclear neutrophils, 55 per cent.

Small lymphocytes, 23 "

Large mononuclears, 16 "

Transitionals, 4 "

Eosinophils, 2 "

DR. PEPPER: Have you seen the feces?

MR. JONES: Not yet.

DR. PEPPER: What does the temperature chart show?

MR. JONES: There is a slight fever, about  $99^{\circ}$  to  $99\frac{1}{2}^{\circ}$  F. The respirations and the pulse are normal.

DR. PEPPER: Now before we ask Mr. Jones for his tentative diagnosis let us briefly re-examine the patient. In the first place, we observe that he does not look very sick, although he does appear somewhat under weight. The yellowness of the scleræ is very obvious, and I feel sure if we look we will see evidences of the jaundice elsewhere. Yes, in this light it is easy to see a yellowish tint to the mucous membrane of his hard palate, and also the palms of his hands seem to be unusually yellow.

Now the enlargement in the neck, as you see, is very evident, and on palpation the mass is clearly composed of a number of enlarged glands somewhat matted together. The individual glands can be felt and vary considerably in size. None show softening, nor does the patient complain of any pain even when I press firmly. As Mr. Jones has told us, the glands involved are those of the anterior cervical chain and the supraclavicular glands. This largest mass lies quite close to the clavicle.

Are these the only glands enlarged, Mr. Jones, or are they part of a general adenopathy?

MR. JONES: I did not find any others.



DR. PEPPER: One should always remember to look for a generalized adenopathy in cases where one or more localized enlargements are found.

It is very easy to overemphasize a marked enlargement of one group and overlook a slight increase in size of other glands.

What other groups should we examine?

MR. JONES: Inguinal, axillary, postcervical, and epitrochlear.

DR. PEPPER: None of these appear to be enlarged unless we consider this very small epitrochlear gland on the left as larger than normal.

Never forget to feel for axillary glands both with the arm extended and with it close to the side, for there are two chains, one superficial to, and one beneath, the axillary fascia. This fascia is relaxed when the arm is to the side and one can then palpate the deep chain.

Now Mr. Jones has omitted two very important groups of glands we should examine for.

SECOND STUDENT: The mediastinal and the mesenteric.

DR. PEPPER: Correct. On percussion I am unable to demonstrate any increased dulness above the heart area or between the shoulder-blades in the gutter of the spine. Remember, however, that this does not exclude enlargement of the mediastinal glands, as we are often unable to demonstrate by physical examination even considerable masses in this region.

Nor can I palpate any masses deep in the midabdomen. I do, however, feel the edge of the liver, especially the right lobe of the organ. It is not tender and the edge does not feel in any way abnormal.

To recapitulate: The physical examination reveals jaundice, anterior cervical and supraclavicular adenopathy, no general adenopathy, a slight enlargement of the liver, especially of the right lobe, with no enlargement of the spleen. Otherwise all is normal and the urine and blood examination are normal except for the presence of bile in the urine and of a moderate degree of secondary anemia.

We now have before us all the facts so far collected. What is your tentative diagnosis, Mr. Jones?

MR. JONES: It seems to me that the jaundice has lasted a long time for a cholangitis, but he has not had any other symptoms of liver disease or of gall-bladder trouble.

DR. PEPPER: Never let yourself be influenced too much by another's diagnosis. The fact that a diagnosis of cholangitis was made six months ago need not influence us. It does tell us, however, that at that time no other cause for jaundice was discovered.

MR. JONES: Well, it seems to me that the most probable diagnosis is chronic cholangitis.

DR. PEPPER: What about the glands in the neck?

MR. JONES: I think they are tuberculous.

DR. PEPPER: Mr. Jones' diagnosis, then, is chronic cholangitis and tuberculosis of the glands of the neck. Let us discuss the question of chronic cholangitis. This diagnosis is a dangerous one to make, and, in general, it is usually wrong. It is a diagnosis which we should only accept after all other causes of jaundice have been excluded. Most of the cases so diagnosed are secondary to cholelithiasis. Acute cholangitis seldom continues to produce jaundice for more than a few weeks, and if the condition persists, doubt is at once cast upon the simple cholangitic nature of the process. Chronic cholangitis due to gall-stones is a result partly of obstruction of the common duct by the stones and partly of ascending infection for which favorable conditions are produced by the presence of the calculus.

Do you think this patient has cholelithiasis?

MR. JONES: No, he has had no colic and he would not be apt to have so much jaundice from gall-stones without some pain.

DR. PEPPER: In what further way does this patient's jaundice differ from that usually seen with gall-stones?

MR. JONES: Too marked and of too long duration.

DR. PEPPER: That is true, but also the jaundice of cholelithiasis is usually intermittent. Remember that this patient states that for almost eight months he has been continuously deeply jaundiced. So let us think of other causes of jaundice which fit in better with this patient's story.

MR. JONES: Complete obstruction of the common duct.

DR. PEPPER: From what cause?

MR. JONES: Carcinoma of the duct or perhaps of the ampulla.

DR. PEPPER: Can anyone suggest additional causes of obstruction of the common duct?

SECOND STUDENT: Worm plugged in the opening.

DR. PEPPER: Yes, that is the type of thing we always remember, but few of us ever see.

THIRD STUDENT: Enlarged glands in the fissure of the liver.

DR. PEPPER: Yes, that is a not very uncommon cause of obstruction. What evidence have we in favor of obstructive jaundice?

MR. JONES: Patient has had the jaundice a long time, it has not shown any intermissions, and it has been intense.

DR. PEPPER: And one more fact which the history mentions, that the patient has noticed his stools to be whitish. This would only occur in more or less completely obstructive jaundice. Let us now leave this side of the case for a few minutes and turn to the other important finding, the enlarged glands in the neck. Why did you think them tuberculous, Mr. Jones?

MR. JONES: The glands are too big to be due to any local infection and there is enlargement on both sides of the neck and no tenderness. And there is no evidence of infection of the tonsils, so if it's not due to local infection, the best diagnosis is tuberculosis.

DR. PEPPER: Are the glands which are enlarged here those which would be enlarged by infection in the tonsils?

MR. JONES: The upper ones are.

DR. PEPPER: We must never forget to have recourse to the facts of anatomy even if we have to look them up in a book. Some of us refrain from referring to text-books from shame of admitting to ourselves that we have forgotten what perhaps we never really knew. The glands which are enlarged in this patient are those of the superficial chain which lies along the sternocleidomastoid muscle and also the deep chain beneath the same muscle. Now what structures do these glands drain?—the ear, back of the head, the tonsils, and, more indirectly, the mouth. The deep chain receives direct drainage from the larynx, trachea,

esophagus, and thyroid. From the fact that there is no enlargement of the submaxillary glands we can safely rule out the teeth and gums as the site of local infection. The absence of swelling of the glands in front of the ear and under the chin is also to be noted. Bilateral enlargement always suggests some other cause than local infection and the absence of tenderness also is important. But having ruled out simple local processes, we are still a long way from a diagnosis of tuberculosis. What other possibilities are there?

MR. JONES: Sarcoma, Hodgkin's disease, and leukemia.

DR. PEPPER: All right; now we must try and choose between tuberculosis, sarcoma, Hodgkin's disease, and leukemia. How can we exclude any of these?

MR. JONES: Leukemia would show in the blood count.

DR. PEPPER: Usually, but not always; during remissions and after treatment the blood-picture may be very nearly normal. However, remissions, even after treatment are, unfortunately, seldom seen in lymphatic leukemia, which is the variety that would suggest itself in this case. But the glandular enlargement of lymphatic leukemia is almost always more general than is present here, and individual chains do not enlarge so prominently. I think we can exclude leukemia.

FOURTH STUDENT: Sarcoma would be more rapid and the glands would not get smaller at any time.

DR. PEPPER: That is true; also sarcoma is often painful and tends to infiltrate the adjoining structures and to fuse one gland to another. What about Hodgkin's disease?

MR. JONES: The enlargement commences in one group of glands.

DR. PEPPER: That is often the case, and it is true that one chain having enlarged, the disease may remain quiescent for months or years, but sooner or later it reappears first with a further enlargement of the chain first affected and then with extension to other glands. This is often, however, not the history of the onset. What groups of glands are commonly the most involved in Hodgkin's disease?

MR. JONES: The anterior cervical.

DR. PEPPER: And in tuberculosis?

MR. JONES: The same glands.

DR. PEPPER: Can we differentiate between the two? Both may be painless and progressive, but the glands of Hodgkin's disease tend to remain discrete, while those of tuberculosis tend to fuse, become adherent to the skin, soften, break down, and even suppurate and discharge. The glands in this patient are very discrete after having been enlarged for a number of months, but we must not have too much confidence, however, in our being able to arrive at a correct diagnosis from the characteristics of the glandular enlargement. I would, however, favor the possibility of Hodgkin's disease.

The fever chart does not help us much. The physical examination does not reveal tuberculosis elsewhere as a help to us, nor is there an enlarged spleen to favor Hodgkin's disease. Nature is not making this diagnosis easy for us.

Now, Mr. Jones, let us return to the jaundice. Will either tuberculosis or Hodgkin's disease explain the jaundice?

MR. JONES: I think jaundice is unusual in tuberculosis, and I do not know about it in Hodgkin's disease.

DR. PEPPER: The liver is frequently enlarged in Hodgkin's disease, and there may be a slight icterus, but deep jaundice in Hodgkin's disease is seldom due to any cause but obstruction of the duct by enlarged glands.

In this patient's case there are two outstanding facts, two prominent or "presenting" symptoms—the jaundice and the enlarged glands. It is always good diagnostic sense to explain by one diagnosis as far as is possible and reasonable everything one case exhibits. Mr. Jones, in his two unrelated diagnoses, has not done this, and, other things being equal, that makes his diagnoses improbable.

Now it does seem possible, without straining probabilities too far, to explain this patient's whole picture with one diagnosis. We have agreed that the jaundice is in all likelihood due to compression of the common duct, and we can at once suggest two possible interrelations with the glands in the neck. One, that the duct is compressed by a neoplastic process of which

the cervical adenopathy is a metastatic manifestation, and the other, that the duct is compressed by enlarged glands, a part of the same adenopathy seen in the neck.

The first of these theories is not very tenable for many reasons. The glands in the neck are not those commonly enlarged secondary to abdominal neoplasm, and the patient's history and condition scarcely those of a neoplasm with such extensive metastasis. What gland, if any, in the cervical region is the site of metastatic enlargement in upper abdominal carcinoma?

STUDENT: The supraclavicular gland on the left side.

DR. PEPPER: Yes, and it is known to many as Ewald's gland. The second theory of relationship is not only plausible, but seems to me to be the correct one to formulate our tentative diagnosis and treatment upon. And of the conditions which might be responsible for this adenopathy we have selected Hodgkin's disease as the most probable.

SECOND STUDENT: Is there any possibility of a syphilitic adenopathy?

DR. PEPPER: A good question. Syphilitic adenopathy is painless, the glands are discrete and hard, but characteristically it does not involve the anterior cervical glands and seldom if ever leads to an enlargement approaching the size of this patient's. So it seems safe to rule out syphilis as a cause of the adenopathy, but, of course, there may be syphilis present, and the routine serologic test will be reported within a day or two.

Are there any other questions?

Well, then, let us adopt as our tentative diagnosis Hodgkin's disease, with an abdominal and cervical adenopathy, and a jaundice due to pressure on the duct by enlarged glands. Have we explained all the patient's symptoms?

MR. JONES: His complaints were constipation and itching.

DR. PEPPER: How would you explain the constipation?

MR. JONES: It is common in jaundice due to obstruction.

DR. PEPPER: Due probably to decreased peristalsis the result of the absence of bile. What about the itching?

MR. JONES: It is very common in jaundice.

DR. PEPPER: That is true, and it may be due to that cause in

this patient, but it is interesting to note that itching is also a not infrequent early symptom in Hodgkin's disease, occurring in perhaps 10 per cent. of cases and often being present in the absence of all demonstrable skin lesions. Now, what investigations would you suggest should be carried out in this case in order to prove or disprove our tentative diagnosis?

MR. JONES: Remove a gland for microscopic examination.

DR. PEPPER: Yes; and what is the histologic picture of Hodgkin's disease, Mr. Jones?

MR. JONES: I'm not sure that I can describe it, but I think there are many eosinophils.

DR. PEPPER: It is difficult to describe. There is always a fine but well-marked fibrosis running between groups of lymphocytic and fibroblastic cells. Often there are scattered endothelial cells, and there may be large numbers of eosinophils, but these may be almost absent and are not an essential part of the picture.

STUDENT: Is there often an eosinophilia in the blood?

DR. PEPPER: Yes, a moderate eosinophilia is seen in at least one-third of the cases at one time or another. An effort has been made to describe a blood-picture characteristic of Hodgkin's disease, but up to the present these efforts have not been very successful, and one should not place much reliance on the blood in making this diagnosis.

Perhaps you should know, however, the views which have been advanced. All agree that a moderate anemia of secondary type is seen and the variance of opinion concerns the leukocytes. A relative or even an actual increase in the mononuclear and transitional cells seems to be the most constant finding, but this is far from being pathognomonic of Hodgkin's disease. The total count of leukocytes may be normal, but there is apt to be a leukocytosis at times, and this recurring leukocytosis is usually due chiefly to an increase in the neutrophilic polymorphonuclear cells. Late in the disease the lymphocytic cells tend to decrease in number. It is interesting that the eosinophilia in the bloodstream does not seem to be proportional to the number of eosinophil cells in the glands. The glands of a case may show enormous



numbers of eosinophils and yet at no time will a blood eosinophilia be noted. As a rule the eosinophilia in the blood does not exceed 10 per cent., but an occasional case is seen with a much higher figure. Perhaps this is an underestimate of the value of the blood-picture as a help in the diagnosis of Hodgkin's disease, but at least it will help to combat the still prevalent impression that this disease should be classed among the "blood diseases," and that it can be diagnosed solely from the blood-picture. It is worth mentioning these opinions and considering the blood count of this patient from their point of view. Mr. Jones found a slight secondary anemia, a low leukocyte count, and in the differential count there was a distinctly high percentage of large mononuclear and transitional cells; 16 per cent. of large mononuclears is a very definitely high figure and deserves emphasis. There is evidence accumulating to favor the opinion that many of these so-called large mononuclear cells of the blood are, in reality, endothelial cells derived from the lining of the smaller blood-vessels. The number of these cells in the blood is said to be increased in typhoid fever, malaria, and measles, as well as in Hodgkin's disease.

Would you institute any treatment at present?

STUDENT: No; it could wait until the gland was removed and examined.

DR. PEPPER: Mr. Jones, do you agree with the tentative diagnosis of Hodgkin's disease?

MR. JONES: I think it is the most probable diagnosis.

DR. PEPPER: We will let it rest there and wait for the histologic examination of the gland. Sections will be shown you as soon as they are obtained.

10/25/1919. DR. PEPPER: Before taking up today's cases let me give you the further details concerning the colored man with jaundice and adenopathy whom we discussed last week.

The question as to whether the jaundice was due to obstruction has been fairly certainly answered by the report that there is no bile pigment in the feces, and that no bile could be recovered from the duodenum even after the introduction into the duo-



denum of 50 c.c. of 25 per cent. magnesium sulphate. The serologic test for syphilis has been negative on two trials, and it was noted that the blood-serum was of a bright greenish-yellow color. The blood coagulation time is slightly prolonged. The phenol-sulphonephthalein elimination is 50 per cent. in two hours.

Last week we discussed and examined for enlargement of mediastinal glands and could not demonstrate any. The Roentgen ray plates of the chest failed to reveal any increase of mediastinal shadow.

We see that there has been no finding which would cause us to alter our reasoning or opinion of last week, and, in fact, the various reports tend rather to confirm our tentative diagnosis.

As most of you know, it was decided to perform an exploratory laparotomy without waiting for the removal and section of one of the glands from the neck.

This seemed wise in order not only to relieve if possible the bile-duct obstruction and the jaundice but also at the same time to make a diagnosis and obtain tissue for study. The majority of you were present when Dr. Deaver operated yesterday. His report of the operation is as follows:

"The gall-bladder is small, thick, and atrophic. The liver and spleen are somewhat enlarged and moderately firm and cirrhotic. A large firm gland is felt at the junction of the cystic and common duct, but as neither duct was dilated above the point of position of gland, probably this was not a source of obstruction to the passage of bile. Other viscera normal. Some retroperitoneal adenopathy along course of aorta. All tissues markedly bile stained."

We think that Dr. Deaver's argument is a good one in view of the long duration of this patient's jaundice, but, on the other hand, no other explanation for the obstruction was found even after careful exploration of the ducts. Dr. Deaver performed a cholecystoduodenostomy and a gland was removed for examination.

The operative findings seem to me to justify our diagnosis and our explanation for the jaundice, although Dr. Deaver's opinion is rather strongly opposed to the latter.

The sections of the gland will be shown you as soon as they are prepared. Frozen sections were not made, as we prefer in this type of case to make a diagnosis only on the best preparations obtainable.

The report on the gland made by Dr. Herbert Fox, received a few days later, was as follows:

Section of the gland removed at operation by Dr. Deaver, 10/23/19. Section shows a marked fibrosis, most pronounced in area of septæ, but also invading. Many round connective-tissue cells. There are very few eosinophils. Aside from fibrosis the most prominent feature is the presence of many large epithelioid cells. Epithelioid cells have round or oval nuclei with rather thin chromatic strands and a definite nucleolus. Epithelioid cells are accumulated here and there in clusters or strands.

Although the picture is not typical of Hodgkin's disease, yet, all things being considered, this diagnosis is the one to be preferred. In this instance, however, it is not justifiable to make a positive diagnosis of Hodgkin's disease from the gland section alone.

## CLINIC OF DR. ARTHUR H. HOPKINS

UNIVERSITY HOSPITAL

### THE TREATMENT OF CATARRHAL JAUNDICE<sup>1</sup>

#### **Etiology; Pathology; Symptomatology; Full Discussion of Treatment with Typical Cases and Results.**

TODAY we will discuss the management of acute catarrhal cholangitis, more commonly called catarrhal jaundice. In order to derive a clearer understanding of the principles of treatment of this condition it will be well to briefly outline the etiology, pathology, and symptomatology of the disease, as it is only by recalling these factors that we are enabled both to form a rational method of procedure and to develop ideas along new lines of therapy. The treatment of the disease to date, though affording relief, has not been very effectual in shortening its duration.

**Etiology.**—First let us discuss the etiology. Certain causes are well established, certain others are not. We know that it occurs more frequently in young, male adults, probably because they are more often subjected to the exciting causes. Gastro-intestinal catarrh due to dietary indiscretion is often a precursor, and is followed by a spreading of catarrh to the biliary papilla, causing swelling of the mucosa and obstruction to the free flow of bile.

Next let us consider the infectious type. We know that it accompanies certain infectious diseases, as malaria, syphilis, typhoid, and pneumonia, and we can readily understand that the infection may travel up into the ducts from the duodenum, producing in its train an inflammatory condition.

<sup>1</sup> Dr. S. Paul Taylor and I have been interested in a new method of treatment which we have developed in the course of the last few months. Dr. Taylor has carried out the technic in detail and it has been largely through his co-operation that it has been possible to present this clinic today.

Again, in portal cirrhosis and cardiac disease with passive congestion of the duodenum and consequent swelling of the mucosa, the papilla too may become congested and obstructed, though we have here also to consider, in the former, radicular cholangitis, in the latter, pressure on the biliary radicles.

You may bear in mind from the above brief etiologic facts that there is one feature which is of the utmost importance, from the viewpoint of therapy, and that is, the very intimate association between alterations in the mucosa of the duodenum and in that of the biliary papilla and ducts. This feature will be recalled during the course of the clinic.

**Pathology.**—Here again may be mentioned swelling, edema, congestion, and increased mucus production at the lower end of the bile duct and of the duodenal mucosa adjacent to the diverticulum of Vater. Swelling alone may obstruct the duct or tenacious mucus may form a plug and thus provoke obstruction, and then succeeding jaundice. It is conceivable that mere catarrhal changes in the mucosa of the duodenum alone, without having spread to the bile passages, may effectually block the flow of bile. The disease, not being fatal, we have but little information based upon autopsies, although "Eppinger, in the report of a case, describes a hyperplasia of lymphoid tissue of the mucosa of that portion of the common biliary duct that runs in the wall of the intestine, which led to complete occlusion of the common duct and dilatation of the rest of the biliary system" (A. O. J. Kelly).

**Symptomatology.**—The early symptoms are usually those of the associated gastro-intestinal catarrh: anorexia, bad taste in the mouth, furred tongue, epigastric distress after food, flatulence, nausea, and at times vomiting. Either constipation or diarrhea may develop; headache and general malaise are usually present.

Jaundice may precede these symptoms, but more often it succeeds them. Usually after a few days of the above symptoms the jaundice gradually develops and becomes more intense, the urine becomes dark and concentrated, and the stools light. Pruritus, slow pulse, etc., are common. Fever is frequently present in the epidemic infectious cases and occasionally in the

other types. The condition runs a course of from three to six weeks or longer. The text-books, in discussing treatment, recommend the symptomatic management of the disease, diet, rest in bed, free water drinking, alkaline waters, bismuth, etc. Calomel followed by a saline aperient is well recommended. As the condition improves, the diet, of course, should become more liberal.

**Literature.**—Like so many of the drugs in our Pharmacopeia, calomel has been the subject of no little dispute with regard to its cholagogic action. Clinicians, as you know, have for years regarded it empirically as a cholagogue, and experimentally its action has been studied by many workers. Among those which have attracted the most attention may be mentioned Scott and also Rutherford and Gamgee, the latter two being members of the Edinburgh Committee. The method employed by these workers was to make biliary fistulæ in dogs, and when the bile regularly escaped from the external orifice, calomel and podophyllin were administered and their effects upon the biliary secretion studied. In dogs thus operated no increase in the flow of bile was noted after calomel. We must not, however, overlook the fact that the conditions under which these studies were performed were not exactly analogous to those in the normal dog, for the animals thus experimented upon died of inanition in spite of nutritious feeding.

Röhrig curarized dogs in which life was sustained by artificial respiration, and then placed a glass tube in the gall-duct so that the bile could escape only through it. Under these circumstances after a time secretion stopped. Calomel rarely re-established the secretion, but its power of increasing and maintaining it beyond the natural time for cessation was very marked.

Rutherford, using the method of Röhrig with some improvements, mixed the drug with bile to facilitate absorption, and injected it directly into the duodenum by means of a hypodermic syringe. He found that calomel had no effect on the biliary secretion, but when to it a minute proportion of corrosive sublimate was added, the effect was very marked. The objection to Rutherford's work lies chiefly in the fact that but two experi-

ments were carried out for each drug used, and at times the results were opposite.

Furthermore, the alimentary tract and digestion of the dog are very different from the human, and hence it is quite conceivable that drugs may affect them very differently. For instance, doses of elaterium that would kill a man can be given to some carnivora without even causing purging.

Stadelman studied the action of calomel on the secretion of bile, also using biliary fistula in dogs, and came to the conclusion that the bile flow was not increased by the administration. Pfaff reports the absence of any cholagogic action of the drug in man in whom a biliary fistula had been made.

Finally, as recently as 1915 Okada repeated the studies of former investigators, using the biliary fistula in dogs, and he, too, concluded that calomel had no influence as a cholagogue.

Notwithstanding the results of these experiments, we wish again to emphasize the fact that the conditions under which they were carried out were abnormal, and to call attention to the fact that the presence of bile in the intestine is a condition necessary to the activity of many drugs.

In view not only of these facts but also of much clinical evidence, many physicians still cling to the earlier conception of the cholagogic action of calomel even though it is based largely on empiricism.

**Treatment.**—Before presenting our cases and discussing their individual treatment we will say a few words with regard to the action of the drugs used.

Meltzer has shown that a 25 per cent. solution of magnesium sulphate, when introduced into the duodenum, will relax the bile duct and permit of a free flow of bile from the gall-bladder to the duodenum. Lyon quite recently reported satisfactory results from the use of this solution in obtaining the duodenal contents for diagnostic purposes, a point which we are ready to confirm, insofar as our studies have been carried.

Our treatment of the cases which we will present to you today is the result of studies we have been carrying out for some time, and in which we have been endeavoring to discover, by means

of the duodenal tube, whether or not an actual increase in the amount of bile secreted does occur in man after the administration of calomel. We are not prepared at this time to present our results, though the findings are suggestive.

We have learned one fact beyond a doubt, which is, that the administration of calomel, 0.095 ( $1\frac{1}{2}$  grain) by mouth does, in many instances, cause a profuse secretion to appear in the duodenum; a secretion which in color, concentration, and general appearance is quite similar to that provoked by the administration of magnesium sulphate, but in volume much greater. At a later date Doctors Taylor, Jonas, and I hope to give you the results of quantitative estimations of the bile content in this secretion, as that is the deciding factor in our studies, as to whether or not calomel increases the secretion of bile.

Today the mechanism of its cathartic action is attributed to its local irritant effect and to increased peristalsis as revealed by the skiagraph. It is believed to act mainly on the duodenum, a point which we can confirm, in so far as its action on that part of the intestinal tract is concerned. Clinically this is supported still further by the fact that in cases of so-called "biliousness," with pale stools, better results are derived more quickly from calomel than from any other drug.

If we claim that it does increase the secretion of the bile, then you may suggest that the "increase in bile is due to the fact that the increased peristalsis causes the bile already in the intestine to be swept out instead of being absorbed." Our answer to that is that "mercurials restore the color of the passages when pale from arrested secretion, often without producing diarrhea, and further, that other and more active purgatives fail to induce the same bilious passages" (H. C. Wood).

CASE I.—The first patient we will show you today is C. T., a student aged twenty-two years. Nine days before he was admitted to the hospital he developed, after exposure to chilling, what he calls an attack of "grippe," this being characterized by slight fever and pains in the back and legs. Three days later his throat became sore, and a day or so after that his friends called

attention to a yellow discoloration of his skin. His bowels were constipated, and after taking blue mass he vomited. The jaundice had gradually become more intense, so that at the end of two weeks it was very marked. He had some generalized abdominal discomfort, especially during the second week. He is usually constipated. His past medical history, aside from a vague intestinal disturbance three years ago and occasional attacks of neuralgic pains, is negative. Social and family history are negative. Physical examination on admission—*i. e.*, nine days after the onset of the present illness—reveals, you will see, a rather poorly nourished young man. His scleræ and skin are deeply jaundiced, and the liver extends from the fourth interspace to 3 cm. below the costal border in the nipple line, and it is distinctly tender to palpation. No other physical abnormalities. Temperature 99° F., pulse 60.

**Treatment: Second Presentation.**—The patient was put to bed and given a liquid diet and calomel (0.065), followed by magnesium sulphate the next morning. We then attempted a new method of treatment which involved the repeated production of a very free secretion from the congested and inflamed duodenal mucosa, and after the production of this secretion, its removal by aspiration. In this way it was hoped that we would be able to lessen the congestion in the region of the ampulla of Vater and permit of a freer flow of bile through the ducts.

The duodenal tube was introduced into the stomach early in the morning, the patient having received no food since the previous night. The gastric residuum when present was withdrawn by aspiration, and then the patient was placed on his right side with the hips elevated and the tube permitted to work over into the duodenum. By siphonage and aspiration the duodenal contents were withdrawn over a period of two hours, at the end of which  $1\frac{1}{2}$  grains of calomel were given by mouth alongside the tube and the siphonage and aspiration continued.

Fifty c.c. of magnesium sulphate (25 per cent. solution) were administered on the second day, as you will see by Table I, to which we will now refer.



TABLE I. CASE I. C. T.

Date.	Amount of duodenal fluid before medication, c.c.	Duration of test in hours.	Drug used.	Amount of fluid after medication, c.c.	Physical characteristics.			Notes.
					Color.	Clear.	Cloudy.	
11/25	50	2	.....	.....	Light yellow	..	+	Jaundice less marked
"		2	Calomel	250	Heavy yellow	..	++	
11/26	180	2	.....	...	Dark yellow	..	+	
"	...	2	Magnesium sulphate, 50 c.c.	330	Heavy yellow	..	+	
11/27	180	1½	.....	...	Light yellow	+		Jaundice still less
"	...	1½	Calomel	250	Heavy yellow	..	+	
11/28	570	4	.....	...	Yellow normal	+	..	
11/29	45	1½	.....	...	Yellow normal	+		
"	...	1½	Sodium phosphate	50	.....	+		
12/2	150	2	.....	...	Yellow	+	..	No jaundice
"	...	1½	Calomel	150	Yellow normal	+		

In the first place you will note that there was only a small flow of bile on 11/25 during the first two hours; then after the administration of calomel there was a much larger amount of duodenal contents recovered. On the succeeding days rather more fluid was recovered both before and after medication. On 11/28 you will note that 570 c.c. was recovered in four hours, with no medication, which would seem to indicate that congestion and obstruction of the bile duct was quite overcome. Two days prior to this the jaundice had become less marked, the urine lighter, and the stools darker, and after this free flow the lessening of the jaundice was very striking. On the 29th sodium phosphate was

administered into the duodenum without appreciable effect. It must also be observed that up to 11/27 the fluid recovered was heavily colored and cloudy, while after that date it was lighter and clear, a point also indicative both of a lessening of the congestion and of a more normal fluidity of the biliary secretion, with unimpeded outlet.

**CASE II.**—The second case we are presenting is one similar in its type of onset to that of the first. This patient, O. L., aged twenty-one, developed one week before admission to the hospital fever, cough, constipation, malaise, pains in the muscles, and headache following exposure to cold. Anorexia, nausea, and vomiting have since developed, and he has noticed in the last three days that his skin has become jaundiced, the urine dark, and the stools light in color. His past medical history is negative, aside from pneumonia in infancy and bronchitis several times. His social and family history are negative.

Physical examination reveals, as you see, a well-nourished, markedly jaundiced young adult with no other abnormalities. Temperature for the past few days has been subnormal, the pulse slow.

**Treatment: Second Presentation.**—Aside from rest in bed and a diet of skimmed milk Table II will illustrate the treatment of this patient.

Here again but little fluid was recovered at first without medication, followed, however, by a larger quantity after the administration of calomel. On the succeeding days, as the calomel and magnesium sulphate exerted their action, we find, as before, a freer flow, and again the fluid becoming lighter and clearer as the treatment progressed. Here too the jaundice steadily decreased, the urine became lighter and free from bile, and the stools darker. It may be well to note that for the first two days after the patient's admission to the hospital we were unable to get the tube into the duodenum. After that there was no difficulty. On the second day of this treatment the symptoms were lessened and by the fourth they were relieved completely.

TABLE II. CASE II. O. L.

Date.	Amount of duodenal fluid before medication, c.c.	Duration of test in hours.	Drug used.	Amount of fluid after medication, c.c.	Notes.
11/12	30	2			
"	...	2	Calomel	380	
11/13	150	2			
"	...	2	Calomel	450	
11/14	100	2	.....	...	Jaundice less marked.
"	...	2	Magnesium sulphate, 50 c.c.	400	
11/15	250	4			
"	...	2	Calomel <sup>1</sup>	0	
11/16	90	2			
"	...	2	Calomel	500	Jaundice markedly lessened.
11/17	...	2	Calomel	380	
11/18	200	2			
11/19	225	2			
11/21	...	...	.....	...	No jaundice.

Bacteriologically the fluid was studied and found to contain many intracellular diplococci, few streptococci, staphylococci, and Gram-positive rods. A mouse was injected, but failed to develop peritonitis. These organisms were all exceedingly resistant to growth on any culture-media, both aerobic and anaerobic.

On 11/15 the calomel was administered directly into the duodenum by way of the tube, and, curiously enough, no action whatever was noted in the course of the next two hours. In the one or two instances in which we administered magnesium sulphate by mouth no increase in the duodenal fluid was provoked.

We therefore assume from our results that the calomel must be given by mouth and the magnesium sulphate by the tube directly into the duodenum if satisfactory results are to be derived.

<sup>1</sup>Introduced directly into duodenum.

**CASE III.**—This patient, A. J., aged twenty, presents a different etiology and onset. For eight weeks prior to his admission he has been troubled with an increasingly severe constipation, the intervals between movements reaching five or six days. Three weeks after onset he noticed that the stools were very light in color. Two weeks later nausea and sour eructations developed, and one week later vomiting and jaundice developed, the latter having now been present for two weeks.

Past medical history: Since infancy he has had gastro-intestinal disturbances, characterized by anorexia, nausea, and constipation at irregular intervals. Social and family history negative.

Physical examination reveals, as you see, a fairly well-nourished young adult with pronounced jaundice. The liver is not enlarged, but there is slight tenderness to pressure over the right hypochondrium and just below the costal border. The urine contains urobilin and bilirubin and the stools are very light in color.

**Second Presentation.**—The treatment, which has been somewhat the same as the others, may be seen in Table III.

The results, as you will see by the table, are somewhat similar to those in the other two cases, but probably owing to the difference in type of case, the duration, and possible chronicity, if we may so term it, the fluid recovered was less in amount both before and after medication. As early as the third day of treatment the stools became dark and the urine lighter in color. By the fourth day there was no bile in the urine. The jaundice steadily lessened, so that by the ninth day of treatment it had disappeared entirely. Cultures were repeatedly made of the fluid, but no growth occurred. Smears showed diplococci and diplobacilli.

One or two cups of tea were given this patient at the time the tube was swallowed, but this seemed to have little effect on the results. During the first few days of treatment there was considerable mucus in the contents withdrawn; this, however, lessened as the patient improved.

The treatment varied somewhat from that of the former two

TABLE III. CASE III. A. J.

Date.	Amount of duodenal fluid before medication, c.c.	Duration of test in hours.	Drug used.	Amount of fluid after medication, c.c.	Physical characteristics.	
					Specific gravity.	Color.
12/16	240	2½	.....	...	.....	Golden yellow
"	...	1½	Magnesium sulphate, 50 c.c.	180	.....	Dark green
"	...	1½	Calomel	95	.....	Light green
12/17	37	1¼	.....	...	.....	Cloudy yellow
"	...	1¾	Magnesium sulphate, 50 c.c.	120	.....	In layers
"	...	1¾	Calomel	90	.....	Dark yellow
12/18	180	1½	.....	...	1.015	Normal greenish
"	...	1¼	Calomel	90	1.015	Greenish
12/19	210	1¼	.....	...	1.031	Yellow
"	...	1¼	Magnesium sulphate, 50 c.c.	127	.....	Green
"	...	1¼	Calomel	120	1.020	Dark green
12/20	60	1½	.....	...	1.014	Light green
"	...	1½	Magnesium sulphate, 50 c.c.	110	1.024	Dark green
"	...	1½	Calomel	60	1.022	Yellowish brown
12/21	60	1¼	.....	...	1.016	Greenish yellow
"	...	1¼	Magnesium sulphate, 50 c.c.	120	1.023	Heavy yellow
"	...	1¼	Calomel	75	1.018	Heavy yellow
12/22	75	1½	.....	...	1.014	Normal
"	...	...	Magnesium sulphate, 50 c.c.	90	1.034	Deep yellow
12/22	...	1½	Calomel	80	1.016	Deep yellow
12/23	68	1¼	.....	...	1.012	Normal
"	...	1¼	Magnesium sulphate, 50 c.c.	150	1.034	Greenish yellow
"	...	1½	Calomel	120	1.014	Normal

cases, in that we used both the magnesium sulphate and calomel throughout, and it will be observed that after the former more fluid was recovered than after the latter, but here we would call attention to the fact that 50 c.c. of the magnesium sulphate was placed directly into the duodenum, and probably the major part of this was withdrawn again by the siphonage and aspiration. This may also account for the rather higher specific gravity of the contents removed after the use of the sulphate. The calomel was administered by mouth, only enough water being given with it to enable it to be easily swallowed.

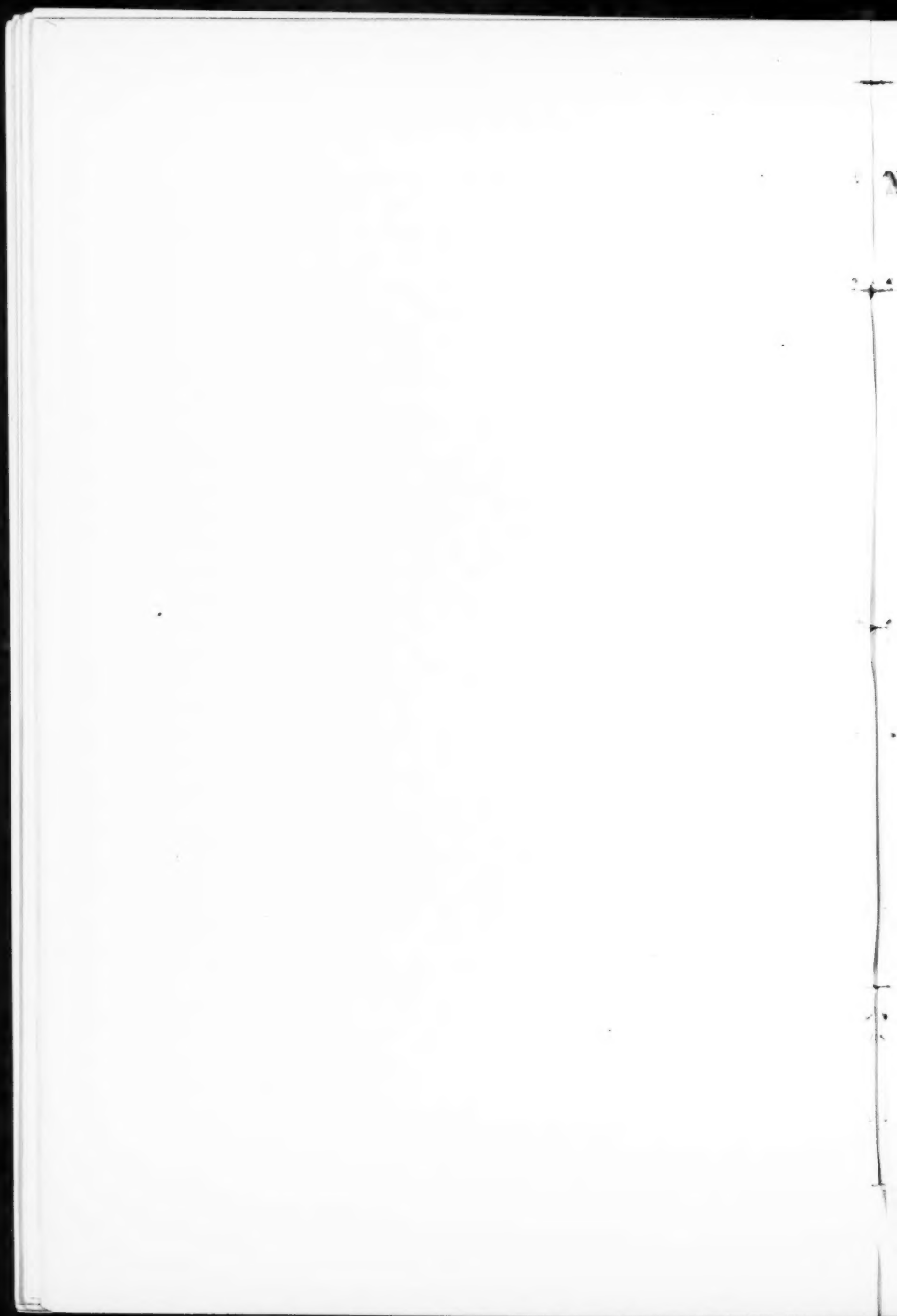
While at present we are unprepared to say definitely that the actual secretion of bile was stimulated in these cases by any cholagogic action of calomel, the marked increase in the duodenal contents of a fluid grossly similar to the usual contents of this organ is suggestive. Certainly its irritant action, insofar as the production of an increased secretion from the duodenal mucosa is concerned, may be considered of much value in lessening the tenacity of the secretion.

In the infectious cases the antiseptic action of the calomel, especially when added to that of the bile, seemed to be most effectual, in that the samples which contained organisms showed them to be incapable of growth in various aërobic and anaërobic culture-media in repeated tests.

Again, the relaxing of the duct by means of the magnesium sulphate seems to be a useful auxiliary measure in attaining rapid results. We believe that if these cases can be thus treated at the beginning of the jaundice much more rapid results may be derived by this method than by any other. Let us cite an instance of this:

There is today a patient in the ward who was admitted a few days ago with the same type of symptoms as those described in the first two cases given above. His jaundice developed on the fourth day of his illness and the following day our treatment was started. Within three days the jaundice disappeared and the symptoms cleared up. The urine, which had been very dark and bile stained, became free of bile, and the stools resumed their normal dark color.

You may raise the objection that the duodenal tube is not a very pleasant form of treatment. We will agree to that, but we will add that neither are the symptoms of catarrhal jaundice very pleasant symptoms, and we find, as a rule, the patient is very glad to have the tube, as he quickly appreciates the alleviation of the symptoms so rapidly accomplished by means of a direct attack at the source of the disturbance.





## CLINIC OF DR. JOSEPH C. DOANE

THE PHILADELPHIA GENERAL HOSPITAL

### DRUG INEBRIETY

Place of Drug Addictions in the Classification of Disease. Not a Disease Entity, but Frequently Only a Symptom of a Deeper Defect. The Manner of Production of the Double Toxemia of Drug and Intestinal Resorption. Etiologic Factors Usually Assigned Only Predisposing in Influence. Relief of Acute Need for Drug Not at All Curative, Since Moral or Mental Pathology Still Exists. Statistics of a Study of 393 Cases. No Secret in Treatment. Necessity of Long-time Commitments, Where Absolute Discipline Can Be Enforced. Ambulatory Treatment Illogic and Illegally Evasive.

#### GENTLEMEN:

It is the purpose of this clinic to invite your attention to some of the underlying factors relative to the use of narcotic drugs for non-curative purposes.

There is a great diversity of opinion as to the place of the drug addictions in the clinical classification of disease. It is held by many that excessive use of opium, or its derivatives, leads to a toxemia which begins and ends with its cause. It is believed by others that drug addiction is but a symptom of a deeper and less easily understood moral or mental defect. It is hoped that we can come to a clearer understanding of the subject by a scrutiny of the histories and symptoms of several type cases.

The first patient, A. C., age thirty-five, gives a history of having used 2 to 14 grains of morphin daily for fourteen years.

He came to this hospital from a southern state, complaining of being nervous and unable to sleep, and was first assigned to the Neurological Service. Later, transfer was made to this

ward. He has tried to "cure himself" four or five times, abstaining from the drug for periods of from eight to ten months. He began the use of drugs from a sense of curiosity, having read DeQuincy, Coleridge, and other writers, who have described the pleasurable sensations arising from opium intoxication.

The personal history of this patient is of some interest. He has followed many and varied occupations, among which may be mentioned mining, railroading, civil engineering, photography, and general mechanics. He attended public school, and later spent some time in a southern academy of good standing. All his life he has been of a studious nature, reading many standard authors. Whenever the particular work in which he happened to be engaged required increased physical or mental strain he suffered with insomnia, and quickly sought recourse to a narcotic.

The family history reveals but few points of interest. Two sisters and one brother are living, but have never been free from "nervousness" and insomnia (patient's statement). One brother suffered with acute mania for four months. Both the father and mother died of diseases incident to old age.

His past medical history is negative.

Physical examination is negative except for a moderate degree of emaciation, and evidences of puncture atrophy on arms and legs.

The outstanding point of interest in this case is the reason assigned for beginning—curiosity, stimulated by reading graphic descriptions of the sensations of a drug inebriate. This is the first patient, in about 2000 coming under my observation, who has specifically assigned this cause for his trouble, although frequent mention is made in medical literature of the harm done to the world by one well-known writing of DeQuincy.

As you know, there are but few drugs which can produce *per se* a diseased condition, which, at least in the patient's mind, the same drugs can relieve. These drugs are continued, and thereby perpetuate the need, which, in turn, is momentarily relieved by further recourse to drug—thus forging an endless chain, encircling and binding the helpless patient.

Have you ever seen a man whose facial expression spells the

deepest misery, his body doubled by abdominal withdrawal pain, and his mental state one of deepest fear and depression, transformed in a twinkling into a self-confident, comfortable, efficient individual, who looks out on life with a desire to live and work? If you have, no further explanation of the reason for continuance is required.

In searching for basic etiologic facts we must not be content to accept at face value the reasons advanced by our patient for the initial step in producing his present toxemia. Rather should we seek the cause for the peculiar individual reaction to the emotions aroused by joy or success, or sadness or failure, which we so frequently see mirrored in a drug user's history. And so in the case before us let us ask, Why did the patient respond as he did to a mere suggestion, gained by reading a word-picture which has been admired by thousands only for its well-balanced beauty of expression? Surely, such a lack of mental and moral poise is not a stable foundation upon which to build a favorable prognosis, even though, as you see, the immediate desire for the drug is now relieved.

The real cause for this patient's trouble lies far deeper than the mere expression of a toxemia which brought him to the hospital. Indeed, a definite neuropathic taint is strongly suggested not only by his inability to long continue in one occupation, but also by his years of insomnia and nervousness.

It should be clearly understood that the term "habit," so frequently used in connection with drug addiction, is misleading and incorrect; for a habit, as you know, is a disposition to do a thing in a certain way, which, made stronger by repetition, each time more closely approaches the involuntary. Mere muscular repetitions do not account for the tendency to continue in narcotism.

Our second patient is of even greater interest from the standpoint of etiology, and illustrates that education and professional training are no barriers to the development of a drug toxemia.

J. K., age sixty-eight, is a dentist by profession. This patient in 1880 began to suffer from right tic douloureux. For seven years his only relief from pain was gained by the use of morphin.

Operation followed operation—fourteen in all—until in 1893 his gasserian ganglion was extirpated. Relief from his symptoms followed, but the need for the drug remained. For the past twenty-four years he has used morphin with but short intermissions.

Physical examination reveals the scars of past surgical interference; vision in right eye practically nil, and moderate senile changes in musculature and cardiovascular system.

In the case under study the blinding, unbearable pain of a neuritis is somewhat explanatory of the beginning of his drug toxemia; and yet we should remember that a neuropathic heredity is thought to be frequently responsible for a neuralgia of the fifth nerve. Twelve years of this double toxemia was sufficient to make life without the narcotic exceedingly difficult.

These etiologic factors would be, therefore, at the outset, in a younger man, very encouraging in making our prognosis. Yet, will power was broken; the judgment, which in consideration of tomorrow, tempers our acts of today, was destroyed, and the very fiber of his moral make-up so degenerated that morphin alone became God.

Allow me by the aid of a third case to illustrate another type of addict:

This boy is eighteen years old; has used drug seven months—12 to 15 grains daily. He left school while in the seventh grade, at the age of fourteen. Occupation, candy vendor at a burlesque theater in this city. He says he began to use heroin because a friend urged him to do so.

Physical examination reveals a rather pale, anemic male; weak face; high arch of palate; hair scanty in axillæ; crines pubes show tendency to form a horizontal line. Skin thin and fair, and a strong suggestion of the rounded contours of the female body exists. Fat over chest and abdominal wall abundant; cardiovascular system negative.

As you will notice, there are here presented evidences of that anomaly known as status thymicolymphaticus. The mental processes are slow, and when not allowed to do as he wishes he becomes childish.

We have not studied enough of these cases for thymic evidences to speak with any certainty, but believe that a larger study would reveal such stigmata as occurring not infrequently.

Contagion from other users is one of the discouraging prognostic factors noted early in the study of many cases. Curiosity often leads the moth too near the flame, and bespeaks but little intelligence and caution on its part. No normal human being blindly eats or drinks an unknown substance without at least questioning the wisdom of such an act.

In a statistical study of 393 patients, covering the four months immediately following the enforcement of the Federal Narcotic Act, we observed many startling facts: 267, or 68 per cent., stated that association with other users was responsible for their narcotism; 71, or 18 per cent., first took an opiate for the relief of pain, while 33, or 8 per cent., blamed—not without a kind of pride—the prescriptions of a physician for their trouble.

It has long been my belief that the occurrence of drug addiction in this type of patient is *more or less accidental*, and that sexual perversion, petty larceny, or any other illegitimate act might as easily be expected. In other words, association with addicts, prolonged medication of unknown content, and doctors' prescriptions are only factors which may somewhat enrich an already more or less fertile and receptive soil. That drug inebriety is frequently only a symptom, and must be considered in that light in outlining our prognosis, seems but a reasonable conclusion. This statement appears to be supported by the study of our fourth patient.

J. S., age twenty-seven, laborer by occupation, has been taking 5 grains of heroin daily (patient's statement).

Addiction began by association with low and dissolute characters. When this patient was asked why he came to us for treatment, he replied that he could not earn enough to buy the drug, and without it he could not work.

Allow me to direct your attention to the fact that there exists an unvarying inverse ratio between the patient's physical ability to work and the amount of money required to keep himself supplied with drug. Ultimately, one of two things must

result. Either the patient is drawn, as this man was, to the hospital for treatment, or money must be procured in one of many illegitimate ways. Indeed, so certain is this conclusion that we are firmly convinced that drug addiction and the frequent outbursts of burglary and daylight robberies have a close connection with drug supply and demand; and that the natural caution of the potentially criminal has been replaced by the momentary recklessness of the narcotized man or woman. Indeed, as we have hinted, the same moral or mental defect which produced the will to steal may have been responsible for the willingness to take the drug. In this hospital not a small percentage of cases coming to us for treatment of drug addiction have criminal records. We cannot but note the frequency with which we observe, in our histories, signs of early delinquency, running away from home to evade school, frequent Juvenile Court probation in males, and early and ill-advised marriage, after scanty schooling, in females.

This latter phase of the etiology is illustrated in our next patient.

This young lady is twenty-three years old and has earned \$15 a week in a book bindery. At the age of fifteen, after reaching the first year in high school, she began frequenting cheap dance halls, and associating with persons who were indifferent toward moral laws. At sixteen she married a drug addict, and finally began using drugs herself. While she knew nothing of the habits or occupation of her husband before marriage, even the fact that although possessing no visible means of support, her husband was apparently well supplied with money did not arouse her suspicions until he was arrested for burglary.

Here we see a young woman who early displayed immoral or, at least, unmoral tendencies, who disobeying and disregarding parental admonitions is now reaping the inevitable reward of those who hold moral laws lightly.

The female addict is probably even more potent in favoring drug contagion than the male, since she so frequently and so easily earns the required funds by sexual promiscuity—thus a difficult and no less hopeless factor is added to the problem.

(In a series of 300 prostitutes, studied by myself at the Philadelphia House of Correction, 40 per cent. were drug addicts.) Moreover, rehabilitation is not favored by her knowledge of the ease of thus earning her living when hard and honest work is urged upon her.

The diagnosis of this condition is not always easy. In the presence of sufficient drug apparent normality is presented. Of course, morphin produces the distinctive pupillary reaction, but heroin does not. Cocain may show perforation of the nasal septum from anesthesia necrosis if inhaled. All may show needle punctures. Thousands of drug users evaded the scrutiny of draft board physicians, and became an inefficient part of our national army.

A diagnosis can only be certainly made after a reasonable period of isolation, with no medication. Withdrawal symptoms, sneezing, yawning, restlessness, vomiting, gastralgia, myalgia, and vasomotor disturbance will surely be observed within a few hours. The prognosis is, unfortunately, in most instances unfavorable, relapses the rule, and cures the exception. If this patient, who, as you see, is comfortable after four days' treatment; who is hungry and has no difficulty in sleeping, may be classed as cured, then our percentage of cures would be very high.

We cannot agree with certain well-known specialists in this line, that it is possible to cure 80 or 85 per cent of our cases. That I have but relieved an urgent and distressing symptom, even as the opiate stops the pain and leaves the gangrenous appendix behind, is, we believe, too frequently the case. The hidden defect still exists, and the drug addict leaves the hospital with the moral or mental pathology undisturbed. Happily, hopeful exceptions exist to this rule, as in morally healthy patients, where only the double drug and autotoxemia exist, the work of the physician is easy indeed.

There is no secret or need for intricate manipulation of drugs in the treatment of this condition. I have shown you 5 patients who have been under our care for four or five days. All are comfortable, and have suffered but little pain or inconvenience during withdrawal. Experience fails to teach us that any of



the belladonna group have any specific action in the treatment of drug toxemia. The action of hyoscin or atropin as delirifacients or depressants of the terminal nerve-endings is valuable in producing more or less painless withdrawal. No specific "unpoisoning" action is believed to exist. Active elimination is indicated, and occasionally support of the cardiovascular system should not be overlooked. More than all else must absolute quarantine be enforced, and the success of your treatment often will lie in forgetting the standing of the patient in the community, and in remembering that the desire for the narcotic will overshadow any other consideration, and that you must combat, with the utmost attention to detail, this almost certain recourse to drug, even under the most favorable circumstances.

In some cities what appears to be a well-intentioned, but misdirected, effort to alleviate the suffering of their patients is being made. I refer to the practice of establishing drug dispensaries, where opiates are dispensed gratuitously in diminishing doses. In a neighboring city 395 addicts applied in one day for this "relief." No solution to the problem seems to lie in this direction, as it is conceded by most informed physicians that the ambulatory treatment of drug toxemia not only is illogic and unscientific, but is illegally evasive.

It is hoped that with increased efficiency in the enforcement of the State and Federal Acts will come a recognition on the part of the authorities that the chief hope for the rehabilitation of most drug inebriates lies in long-time commitments to an institution where discipline can be enforced; and where long-forgotten respect for honest work and workmen can be reinculcated. The drug user while under commitment could at least be self-supporting, and the chance for permanent relief would be somewhat in proportion to the length of his incarceration.

Finally, as in few other conditions will you have occasion to display not only that unfailing tact and untiring patience, which is so essential in your profession, but also to command and hold your patient's confidence in your ability to bring him safely through a difficult and fearsome illness.



## CLINIC OF DR. CLIFFORD B. FARR

### POLYCLINIC HOSPITAL

---

#### PAINLESS GASTRIC CRISES

##### GENTLEMEN:

The patient before you, whom some of you saw previously in the x-ray room, presents a combination of symptoms and signs which serve as a nice criterion of the thoroughness of our methods of examination rather than of our diagnostic skill. The diagnosis is evident if a thorough routine examination has been carried out, but may be as easily missed if this has been slighted. In the Gastro-intestinal Clinic we have used a rather elaborate outline for recording cases, but have found that the mass of detail is likely to overshadow the really important symptoms and signs. For this reason I believe it is best in studying our cases to be guided by the patient's chief complaint and by the physical signs as they present themselves, and then afterward to check off on a separate printed sheet, arranged as far as possible in tabular form, routine observations which, while chiefly of negative value, occasionally give the clue to the diagnosis (*e. g.*, character of pulse, glandular enlargements, eruptions, reflexes, herniæ, etc.). The record in the present case, I may say, is not an example of such a plan.

Our patient, John T. L., a colored policeman (special), thirty-seven years of age, was admitted to my service at the Polyclinic Hospital on November 9, 1919, complaining of *pain in the "pit" of the stomach, nausea, and persistent vomiting*. Six years ago (1913) the patient for the first time suffered from an attack similar to the present, which lasted three weeks. This he attributed to the eating of a "cold stew." The pain

at that time started in the midepigastrum and radiated to the right flank. The patient vomited almost continuously and was unable to take any food. The vomitus was of a green color; at no time did it contain any blood. Following this primary attack he was perfectly well for about seven months, when he was again seized with pain in the epigastric and umbilical regions, followed by vomiting of greenish material. He had two similar attacks in 1918 and again in June of the present year. On the latter occasion he was admitted to this hospital and was treated for "indigestion," from which he says he did not entirely recover, so that any heavy meal disagreed with him. On consulting the hospital records we find that he was admitted June 12th and was discharged June 21, 1919, with the notation: "Acute gastritis, cured." His chief complaint was nausea and vomiting. The trouble had begun two days before admission, and he had been unable to keep anything on his stomach. No other symptoms except discomfort from the continual vomiting and retching. The examination was negative except for rigidity of the epigastrum. Night temperature for eight days ranged from 99° to 99½° F. The nurse noted constant hiccup, vomiting of brownish material, and soreness in the abdomen. Vomiting last recorded on the 18th, one week from the onset. (No distinct pain was recorded.) The urine was negative. Wassermann reaction moderately positive.

Gastric contents after an Ewald meal: Quantity, 120 c.c., green; total acidity 42; combined 19; free HCl 12, acid salts 11; no lactic acid. No occult blood. No Oppler-Boas bacilli.

The present attack began shortly prior to admission, with pain in the "pit of the stomach," followed by vomiting and retching. Subsequently, on being asked to indicate the "pit of the stomach," he pointed to the hypogastrum. "In one of my cases the attacks always commenced with pain in the region of the bladder." The pain was never marked. The vomitus was rather green, but at no time did it contain any blood. He said that his appetite was habitually poor, and that he suffered from constipation from time to time. He stated that he had

<sup>1</sup> Oppenheim, T. B., *Nerv. Dis.*, English trans., 1911, vol 1, p. 153.

had six attacks at varying intervals (as described above), and that the attacks were increasing in severity. There was some persistence of gastric disturbance in the free intervals.

The patient is a heavily built, middle-aged negro, with heavy features and prominent jaws. He appears somewhat drowsy, but answers questions clearly and intelligently. The chest is well formed, the expansion is good, resonance normal, breath sounds clear, no râles. The heart area is normal; the heart sounds are rather weak or distant. The abdomen is rounded and the muscles well developed. There are no palpable masses or organs; there are no areas of tenderness. The stomach dulness after the ingestion of considerable fluid extends two fingers below the umbilicus. We are unable to outline it by succussion splash. Seen in the fluoroscope after the first barium meal, you noticed that the stomach appeared to be dilated and the pyloric end rounded off, the duodenal cap being absent. The epitrochlear glands are enlarged, the pupils react very sluggishly to light. The patellar reflexes are also very much diminished. We observed in the x-ray room that the patient walked with difficulty and could not stand unsupported in the dark. Romberg's sign tested in the usual way is positive, but he performs the finger-nose test fairly well. He states that he had no difficulty walking in the dark prior to admission, but admits that he was subject to the "blind staggers." Dr. J. H. W. Rhein has gone over his nervous symptoms at my request, and has made the following observations (condensed): Marked Argyll-Robertson pupil; no facial paralysis; no fifth nerve involvement; finger-to-nose test normal; biceps jerks equal and active; no history of lancinating pains in legs or arms; slight lateral sway with eyes closed and feet together. Knee-jerks absent; Babinski absent; pain sense preserved in legs; sense of touch in legs not delayed; appreciation of light touch diminished in both thighs; muscular sense in legs normal; sphincters controlled; no loss of sexual power; no joint lesions.

Between November 10th and today (Nov. 18th) the temperature ranged from 98° or 99° to 100° F., and the pulse from 85.5 to 105. There was no disturbance of respiration. (Subse-

quently until his discharge on the 25th, temperature, pulse, and respiration were normal.) During the early stages vomiting of dark green fluid and hiccup recurred many times during the night as well as during the day. The bowels could be moved by enemas at all times. By the seventh day of the attack vomiting had ceased and dyspeptic symptoms were rapidly clearing up.

*Laboratory Reports.*—Three examinations were made of the urine. The specific gravity ranged from 1022 to 1026; the reaction was alkaline in all specimens. No albumin was present, no sugar, no casts. There were occasional white blood-cells, bladder cells, cylindroids and phosphates, amorphous or crystalline.

On the day of admission there were 4,700,000 red cells, 9280 white cells, 95 per cent. of hemoglobin.

A Wassermann reaction taken on November 13th showed a 4+ reaction.

Sputum, no acid-free organisms were found.

Stomach contents extracted during the attack (Nov. 11th): Occult blood positive; lactic acid negative; no Boas-Oppler bacilli. Total acidity 70; Free HCl 33. (The patient insisted on leaving before a "fractional" study could be made in the "interval.")

The resident physician attempted lumbar puncture, but met with difficulties, and desisted. As the diagnosis seemed sufficiently clear, the attempt has not been repeated.

The following is the report of the x-ray observations on Nov. 12th to 14th (previously alluded to): "I am not able to see the duodenum fill up by fluoroscopic examination. The stomach emptied itself in normal time, but there was a temporary gastrectasis; there was unquestionable stasis."

The treatment during the attack was symptomatic and palliative; absolute confinement in bed, liquid diet, bismuth, ice to the abdomen, enemata, etc. Now that the acute symptoms have subsided, he has been placed on mercury by mouth and inunction and on weekly injections of arsphenamine.

Were it not for the nervous symptoms—Argyll-Robertson

pupil, positive Romberg sign, loss of patellar reflexes, and the positive Wassermann reaction, etc.—we might be led into a discussion of the differential diagnosis, but with these findings, the diagnosis of *tabes dorsalis* with gastric crises is hardly in doubt.

It is true that the symptoms do not coincide exactly with Charcot's classical description of gastric crises, but if we consult Gull,<sup>1</sup> to whom Charcot assigns the credit of the first definite recognition of this complication (in a case found at autopsy to have degeneration of the posterior columns), we find manifestations comparable to those in our own case. His patient had *repeated attacks of vomiting lasting for many days, uninfluenced by any remedies. The vomited matter was copious, greenish, and mucous. The bowels continued to act freely with relief to the sickness. The irritability of the stomach was attributed to the state of the cord.* While the diagnosis is usually easy if the cord lesion has advanced sufficiently to cause definite changes in the reflexes, the possibility of error is still present, for, curiously enough, Gull's patient finally died of intestinal obstruction due to *volvulus*, with symptoms not unlike those attributed to the cord lesion. In many instances gastric crises are the initial symptom in *tabes*.

Cases of *tabes dorsalis* with painless gastric crises, of which our patient is an example, while unusual, are occasionally seen. We had an even more typical case under observation at the Philadelphia General Hospital (service of Dr. David Riesman) for more than a year and a half.<sup>2</sup> The latter patient had his first attack of vomiting in July, 1914, more than a year before his first admission, but even as late as August, 1915 a very competent oculist reported that his "pupils were equal and responded promptly to light." He denied venereal disease and his reflexes were noted as present. He was a heavy drinker, so that a diagnosis of alcoholic gastritis was made at that time. The

<sup>1</sup> Guy's Hosp. Reports, 1858, 3d S., vol. iv, 169.

<sup>2</sup> This patient was shown at the Section on General Medicine of the College of Physicians on October 25, 1915, by Dr. S. V. Emerson, the intern on service.

gastric analysis at a somewhat later date showed a total acidity of 100, and free HCl of 60. His vomiting attacks recurred every few weeks or months, and he gradually developed definite evidences of tabes: sluggish pupils, absent knee-jerks, ataxia, etc. The Wasserman reaction was positive. He finally died during an attack June 5, 1917. At this time the following note (edited) was made by the resident physician, Dr. Kendall: "The patient has suffered from periodic attacks of vomiting, very severe in type, with distressing nausea and hiccup, but no pain. The attacks have ordinarily cleared up after a few days and the patient has been able to resume work in the hospital office. No medication seemed to shorten the course of his attacks. He complained at times of shooting pains in the legs. On the first of this month he was admitted to Ward 14 in one of these attacks. Nothing seemed to relieve him and the vomiting became almost continuous. On the 3d he became very delirious; on the following day had four convulsions, and died on the 5th."

Charcot (1880)<sup>1</sup> describes a typical gastric crisis as follows: "Suddenly, most often at a period when lightning pains are affecting the limbs, the patient complains of pains which, beginning in the loins, appear to rise on each side of the abdomen. Simultaneously he complains of pain between the shoulders and radiating around the base of the trunk. . . . Vomiting, almost incessant and extremely painful, is often associated with gastric crises. The ingested food is first rejected, then a colorless mucoid liquid sometimes mingled with bile or tinged with blood. . . . The gastric crises . . . generally persist, just as do the crises of lancinating pain almost without respite for two or three days, and it is very remarkable that in the intervals between the attacks the functions of the stomach are generally carried out in a very regular manner."

Gentlemen, the cases whose histories I have presented to you, while evidently of the same nature as those pictured so vividly by Charcot, differ radically in that, while his cases usually had vomiting in addition to the typical painful sensations, our cases

<sup>1</sup> *Leçons sur les Maladies du Système Nerveux*, Tome iv, 3 ième ed. Par., 1880.

were chiefly remarkable for the severity of the vomiting, and only secondarily or not at all for the pain. Here again recent French authors,<sup>1</sup> with the national facility for analysis, come to our aid by outlining several types of tabetic crises:

First, crises of the motor type, or vomiting crises.

Second, crises of the sensory type, or painful crises.

Third, crises of the secretory type, or tabetic gastrosuccorhea.

Our authors go further, if a little dubiously, and define a vasomotor type, causing hemorrhage, but here we hesitate to follow them. In this connection Dr. Frazier<sup>2</sup> states that crises of the motor type are due to involvement of the *vagus*, while the more typical sensory variety is referable to the sympathetic fibers (splanchnics) derived from the *posterior thoracic roots*.

Finally, we must gather the scattered threads of our discourse and add a brief note on the treatment. We have, then, a case with gastric crises of the motor type, occurring in the early stages of tabes, and on this account very liable to be mistaken for gastritis, appendicitis, gall-stone-dyspepsia, gastric ulcer, gastrosuccorhea, etc. The important point is to be on the lookout for tabes whenever unexplained vomiting or gastralgia are in question. The atypical forms without severe gastralgic pains or lancinating pains in the extremities are peculiarly puzzling. In the case seen at the Philadelphia Hospital the pupillary and deep reflexes were very variable and seemed to be more abnormal during the seizures and to improve in the intervals.<sup>3</sup>

The treatment of the attack is purely symptomatic and usually futile. It is suggestive that immediately after a severe

<sup>1</sup> Carnot and Bruyère, Bull. et Mem. de la Soc. Méd. des Hôp., 1917, 41, 1103.

<sup>2</sup> Frazier, Surgery of the Spine and Spinal Cord, N. Y., 1918, p. 695. (Contains an excellent discussion of the medical and surgical aspects of gastric crises.)

<sup>3</sup> Oppenheim, loc. cit., p. 145: "In isolated cases the reflex immobility of the pupils has been found to be intermittent (Eichorst, Treupel, Mantoux), i. e., it may be present only for a time and then disappear." On the other hand, I am informed by competent oculists and neurologists that the Argyll-Robertson and Westphal phenomena, once fully developed, do not recede.

attack the patient may return to a normal diet. For patients like our own, in the early stages of tabes, intense specific treatment is indicated. Operation, consisting of section of the posterior roots, as suggested by Prof. Förster of Breslau,<sup>1</sup> has been successfully employed for painful crises. Prof. Förster considers that cases without pain should be regarded as unsuitable for this procedure.

More recently section of the vagus below the diaphragm (Exner) or near its origin in the medulla (Kuettner) has been proposed for the motor type.<sup>2</sup>

<sup>1</sup> Lancet, 1911, ii, 76.

<sup>2</sup> Frazier, *loc. cit.*



